

Types of Cells in the heart :-

- ① Automatic Cells
- ② Normal Cells

Heart has 3 types of Automatic Cells
"Pace Maker"

(القلب يسرع كل م الأسرع)

① SAN	الوسع	60 ~ 100
② AVN		40 ~ 60
③ Purkinje fibers		30 ~ 40

AVN • ① accessory Pace maker

② physiological delay (to allow filling)

③ protect the Ventricle (No more than 150 impulse can pass)

④ "e.g. from arrhythmogenic focus in atrium"

* ④ One way Gate "No retrograde Conduction"

Atrioventricular Dissociation :-

① Complete A-V Block

② arrhythmogenic focus in Ventricle (Ventricular tachycardia)

NB Atrial flutter is not dissociation (the heart follow 1 focus in atrium)

NB Any management: I - diagnosis

- C/P
 - symptoms
 - signs
 - Complications
- investigations
- D.D.

II - treatment

NB To fulfill tissue Demand :-

- Contract** **Relax**
- ① Patent Circulation (No Block) (Aortic or pulmonary Stenosis)
 - ② Normal Volume (no Volume overload; Regurge, Hyperdynamic Circulation)
 - ③ Normal Contractility (systole) (Myocarditis, infarction)
 - ④ Normal filling (Huge pericardial effusion, Cardiac tamponade)

NB HF

- Low CO HF → more Common
- High CO HF → ~~more Common~~ Hyperdynamic Circulation

VISIT...

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HEART FAILURE

DEFINITION

كلى
Clinically

It is a CLINICAL SYNDROME resulting from inability of the heart to pump enough blood sufficient to meet the metabolic needs of the body.

ETIOLOGY

I. LEFT - SIDED HEART FAILURE

1. Left atrial failure:

- ◇ Mitral valve disease: MS. or MR
- ◇ Left atrial myxoma: rare cause.

2. Left ventricular failure:

◇ Pressure overload:

- SYSTEMIC HYPERTENSION (تفاديس)
- Valvular disease: Aortic stenosis.
- Congenital disease: Coarctation of aorta. (descending)

◇ Volume overload:

- HYPERDYNAMIC CIRCULATION. { Anemia
Thyrotoxicosis
- Valvular disease: Aortic regurge & Mitral regurge.
- Congenital disease: e.g. VSD

◇ Impaired cardiac contractility:

- CAD: Myocardial infarction
- CardioMyopathy. (DCH)
- Myocarditis.

Segmental disease

Global disease

◇ Impaired cardiac filling:

- Pericardial disease: Cardiac tamponade, Constrictive pericarditis.
- Myocardial disease: (RCM.)

* Hyper Dynamic Circulation :-

* Anemia : ① $\downarrow$ Hb $\rightarrow$ $\downarrow$ Viscosity $\rightarrow$ $\uparrow$ Velocity

② Hypoxia $\rightarrow$ Severe VD "accumulation of lactic & pyruvic acid"

* Thyrotoxicosis : ① $\uparrow$ T_3, T_4 $\rightarrow$ +ve inotropic & +ve chronotropic

② $\uparrow$ needs "due to $\uparrow$ BMR"

why LVF $\rightarrow$ RVF? "Most Common Cause"

LVF $\rightarrow$ Back Pressure $\rightarrow$ Pulmonary Congestion $\rightarrow$ reflex pulm. VC in arteries
 $\downarrow$ Pulm. HTN.
Pressure overload on R.V.

why COPD $\rightarrow$ RVF? "2nd Common Cause"

COPD $\rightarrow$ $\downarrow$ O_2 $\begin{cases} \rightarrow$ VD. systemic \\ \rightarrow V.C. pulmonary $\rightarrow$ pulmonary HTN $\rightarrow$ Pr. overload on R.V. \end{cases}

LSHF

RSHF

LAF: - Ms
- Lt Atrial Myxoma

LVF: $\left\{ \begin{array}{l} \text{CAD} \\ \text{syst HTN} \end{array} \right.$

I - Pressure Overload

- ① Systemic HTN
- ② Congenital: ~~aortic~~ ~~SS~~ Coarctation
- ③ Valvular: AS

II - Volume Overload

- ① Hyperdynamic Circ
- ② Cong.: VSD
- ③ Valvular: MR, AR

III - ↓ Contractility

- ① CAD (MI) segmental
- ② Myocarditis
- ③ Cardiomyopathy } Global

IV - ↓ Filling

- ① Pericardial [tamponade, Constrictive pericarditis
- ② RCM

RAF: - TS ?
- Rt. atrial Myxoma

RVF: $\left\{ \begin{array}{l} \text{LVF} \\ \text{Pulm disease} \end{array} \right.$

I Pressure overload

① Pulm HTN (COPD, LVF, Acute PE)

- ② Congenital: PS
- ③ Valvular: PS

II Volume Overload

- ① Hyperdynamic Circ.
- ② Cong: ASD
- ③ Valvular: TR, PR

III - ↓ Contractility

- ① CAD (MI) segmental
- ② Myocarditis
- ③ Cardiomyopathy } Global

IV - ↓ Filling

- ① Pericardial [Cardiac tamponade, Constrictive Pericarditis
- ② RCM

Etiology + Precipitating Factors

2I³ 3A 3P

II. RIGHT – SIDED HEART FAILURE

1. Right atrial failure:

- ◇ Tricuspid valve disease: Tricuspid stenosis.
- ◇ Right atrial myxoma: very rare cause.

2. Right ventricular failure:

◇ Pressure overload:

• PULMONARY HYPERTENSION:

- Handwritten notes: "just RHF" with an arrow pointing to this section.*
- chronic → LVF (the most common cause of RVF).
 - Pulmonary disease, e.g. COPD (cor – pulmonale). *Handwritten: "لانسى"*
 - acute → Acute pulmonary embolism (causes acute RVF).

- Valvular disease: Pulmonary stenosis. (*Handwritten: "Rheumatic"*)
- Congenital disease: Pulmonary stenosis.

◇ Volume overload:

- HYPERDYNAMIC CIRCULATION.
- Valvular disease: Pulmonary regurge & Tricuspid regurge.
- Congenital disease: e.g. ASD (*Handwritten: "why not VSD" and "لانسى السكتة"*)

◇ Impaired cardiac contractility: (*Handwritten: "during diastole"*)

- CAD: Myocardial infarction Segmental disease
- *"dilated"* CardioMyopathy. (*Handwritten: "DCM"*) Global disease
- Myocarditis.

◇ Impaired cardiac filling:

- Pericardial disease: Cardiac tamponade, Constrictive pericarditis.
- Myocardial disease: RCM.

- The most common causes of LVF are:

- CAD.
- SYSTEMIC HYPERTENSION.

- The most common causes of RVF are:

- LVF.
- PULMONARY DISEASE.

write
e 4
Causes of HF
Etiology

Compensated

decompensated

PRECIPITATING FACTORS

2I + 3A + 3P
LC
DE

- In a patient with compensated HF, these factors may decompensate the heart.
- During ttt of HF, if these factors are not removed, HF will not improve (**refractory HF**).

الأسباب

① ♥ **Infections:** esp. infective endocarditis, rheumatic activity & chest infection.

② ♥ **Iatrogenic:**

- Corticosteroids & Calcium - channel blockers.
- Sudden ○ Discontinuation of anti-failure treatment. "must treat Cause"
- Excessive salt intake & IV fluids.

③ ♥ **Acute myocardial infarction.**

④ ♥ **Arrhythmias.** (The most Common Cause of Arrhythmia is ISCHEMIA = CAD)

⑤ ♥ **Anemia, thyrotoxicosis & other causes of hyperdynamic circulation.**
- Volume overload → ↑HR, ↑work → acute cases - ↑needs

⑥ ♥ **Physical & emotional stress.** } ↑tissue needs, ↑Cortisol

⑦ ♥ **Pregnancy & late labor.**

⑧ ♥ **Pulmonary embolism.** → ↓bl. from Lung to Heart → ↓CO

PATHOPHYSIOLOGY

1. The clinical manifestations of HF result from:

low CO
Congestion

► **Forward Failure:** Failure of the heart to eject a sufficient CO:

- This gives rise to manifestations of **low CO**.

► **Backward Failure:** Failure of the heart to accept the VR:

- This gives rise to manifestations of **congestion**.

○ **Pulmonary congestion:** in case of Left - sided HF.

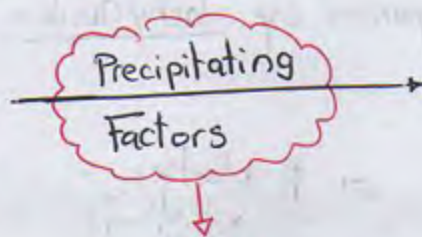
○ **Systemic congestion:** in case of Right - sided HF.

NB

RSHF = Low CO + Systemic Congestion

LSHF = Low CO + pulmonary Congestion

Compensated HF
= Controlled
= not Symptomatic

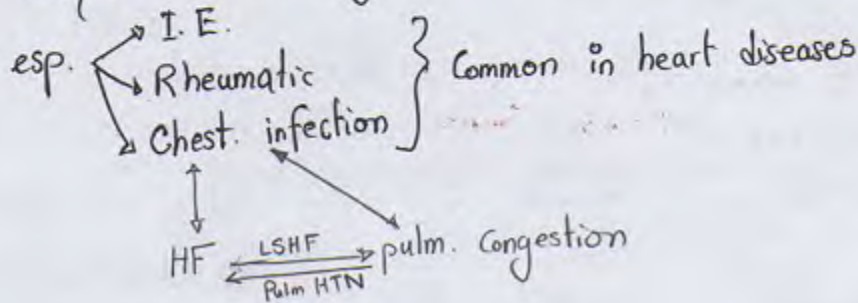


Decompensated
= uncontrolled
= symptomatic

- ① All are Acute Factors
- ② ↑↑ work of the heart → exhausted
- ± ③ ↑↑ needs of tissues (i.e. ↑ BMR)

Refractory HF = Not Responding to the usual tt of heart failure
Persistent refractory HF is due to untreated precipitating factor

Infections → is the most Common Cause
Any where in the body, infection will affect the heart



Iatrogenic

* **Cortisol** is **Contra indicated** "aldosterone like effect" → salt & water retention
↳ volume overload

We treat it ~~Diuretics~~ **diuretics**

* **CCB** is Contra indicated → -ve inotropic → ↓ Contractility

We treat it **Digitalis**

* CAD, HTN are treated by **CCB**

↳ if lead to **HF**, **CCB** become Contra indicated.

* Salt & intake

نسيب تكتب في اليوم: diet ال دكتور
مبعضش الكلام: عيب عيان

Acute Myocardial infarction: CAD

Most Common Cause of arrhythmia
is **ISCHEMIA**

* Hypertensive HF $\xrightarrow{\text{acute MI}}$ uncontrolled.

* CAD $\xrightarrow{\text{Cause}}$ HF $\rightarrow$ controlled $\xrightarrow{\text{Arrhythmia}}$ uncontrolled HF

- ↑ HR "300/min"
- acute

لرئسي

Heart failure is always accompanied by "Tachycardia"

Except in Some Cases :-

① Drug : Digitalis or β -blocker
Not diuretics Not ACE-I

② Disease : A-V nodal block

2. Compensatory mechanisms (Cardiac reserve):

- These mechanisms try to restore the cardiac output.
- They are beneficial within limits.
- If they exceed these limits, they will aggravate HF. (become harmful)

► TACHYCARDIA: $> 100 \text{ beat/min}$

1. Mechanism: sympathetic stimulation mediated by:

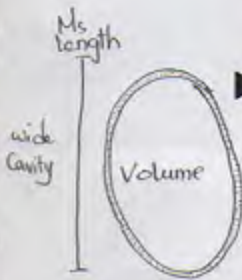
Medulla
↓
HR
Atrial stretch receptors

- Marey's law: HR is inversely proportional to BP.

- Bainbridge reflex: Increased RA pressure causes increased HR. (right atrium)

2. Effect:

- It increases the CO.
- If it is marked ($> 160 / \text{min}$):
it will ↓ the diastole, ↓ the ventricular filling & thus ↓ the CO.



► DILATATION:

- Increased LENGTH of cardiac muscle fibre: in VOLUME overload.
- It increases the force of contraction (according to Starling's law).
- If it exceeds certain limits, it will lead to diminished contraction.

normal
Cavity



► HYPERTROPHY:

- Increased THICKNESS of cardiac muscle fibre: in PRESSURE overload.
- It increases the force of contraction. (Ms bulk increase)
- If it exceeds certain limits, the hypertrophied muscle cannot get its adequate blood supply (becomes ischemic) → diminished contraction.

normal
or
reduced
Cavity



► REDISTRIBUTION OF BLOOD FLOW: the only mechanism that never become harmful

- There is diversion of blood: reflex spontaneous autoregulation: autonomic nervous system (ANS)
 - From less vital organs (e.g. skin & muscles),
 - To more vital organs (e.g. brain & heart).



► HYPERVOLEMIA:

(due to salt & water retention) (↓ GFR)

- It increases the preload & force of contraction.
- If it exceeds certain limits, it will aggravate HF.

Pathophysiology of Heart Failure

I C/P of HF due to

Backwards failure

- failure to accept VR
- symptoms of Congestion

Forwards failure

- failure to Eject enough CO
- Low CO sympt.

II Compensatory Mech "Cardiac Reserve"

Mech to restore CO
Beneficial within limits $\xrightarrow{\text{if exceed}}$ aggravate HF

↑HR

Mech: Symp. mediated by:

- ① Marey's law ($HR \propto \frac{1}{BP}$)
- ② Bainbridge ($\uparrow R.A.P. \rightarrow \uparrow HR$)

Effect

- ✓: ↑CO
- X: if >160
↓ diastole → ↓ filling
→ ↓CO

Hypertrophy

Mech

↑ Ms thickness
in R. overload

Effect

- ✓: ↑ Contractility
- X: if exceed certain limit → ischemic
→ ↓ contraction

Dilatation

Mech

↑ fibers Length
in Volume overload

Effect

- ✓: ↑ Contractility (Starling's)
- X: if exceed certain limit →
↓ Contractility



Redistribution

Mech

VC → skin, Ms
VD → heart, Brain

Effect

- ✓: divert bl. from less vital to more vital organs

HyperVolemia

Mech

salt & water retention
(↓GFR)

Effect

- ✓: ↑ preload
→ ↑ Contractility
- X: if exceeds → worsen HF

III Neurohormonal Changes

RAAS

* Beneficial:

- - Maintain Bl. P.
- * - ↑ perfusion to vital organs

* Harmful:

- * Salt & H₂O retention (↑ Preload)
- Potent VC (↑ after load)

SNS

* Beneficial:

- ↑ Contractility (+ve inotropic)

* Harmful:

- VC → ↑ afterload (Pressure overload)
- Arrhythmogenic (↑ excitability)
- direct Myocardial damage (long term)



ANP

* Beneficial:

- Natriuresis
- Peripheral VD (A & V)

IV Asynchronous Vent. Contraction

- in 30% of DCM there's "intra Ventricular Conduction delay" or BBB leading to
 - (A) The 2 Vent. beat slightly out of phase
 - (B) Segmental a-synchrony within LV may occur
- } → ↓ Efficiency of Vent. in HF

ACE-I

⇒ dilate Veins & Arteries

1st Choice in $\uparrow$ of HF

(NB) if Dry Cough → give ARB "Angiotensin Receptor Blocker"

(NB) Catecholamine of SNS

Normal Cells $\xrightarrow{\text{increase excitability}}$ Automatic Like Cells

* But: direct Myocardial damage on Long term (chronic patient)

SO: β -Blockers are given to the chronic patient

2nd Choice in $\uparrow$ of HF

Diuretics

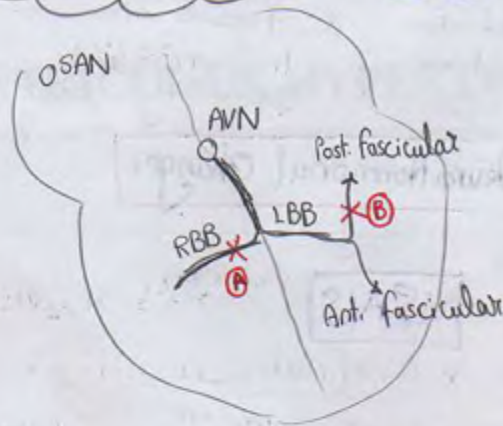
3rd Choice

Digitalis

4th Choice

Asynchronous Vent. Conduct²

In Dilated
Cardiomyopathy
30%



Not Physiological
Pathological

3. Neurohormonal changes in HF:



Some of these changes are beneficial, others are harmful:

- a) Activation of the RAAS (Renin - Angiotensin - Aldosterone System)
- Beneficial effect: maintains BP & perfusion of vital organs.
 - Harmful effects:
 - The increased angiotensin II: results in vasoconstriction. → *Pr. overload*
 - The increased aldosterone: results in salt & water retention. → *Volume overload*
- b) Activation of the SNS "Sympathetic = Catecholamine"
- Beneficial effect: increases the ventricular contractility.
 - Harmful effects:
 - Increases the afterload. (VC.)
 - Induces arrhythmias. (due to increased excitability)
 - Causes direct myocardial damage. (on the long term: months to years)
- c) The Natriuretic peptide system (ANP) Atrial natriuretic Peptide
- From atrium mainly But also from Rt Vent.*
- HF → Stagnant bl. in atrium → stretch atrial wall → ANP → ↓ after load & pre load*
- ④ loss of Na⁺ from renal tubule*

4. Asynchronous ventricular contraction: *Very Recent*

- ① In approximately 30% of patients with heart failure due to *dilated* cardiomyopathy: *"DCM"*
- There is an abnormality in the heart's electrical conducting system (called an "intraventricular conduction delay" or bundle branch block).
- ② This problem causes the 2 ventricles to beat in an Asynchronous fashion: therefore, instead of beating simultaneously:
- Ⓐ ○ The 2 ventricles beat slightly out of phase.
 - Ⓑ ○ Also: segmental asynchrony within the LV may occur.
- This asynchrony greatly reduces the efficiency of the ventricles in patients with heart failure, whose hearts are already damaged.

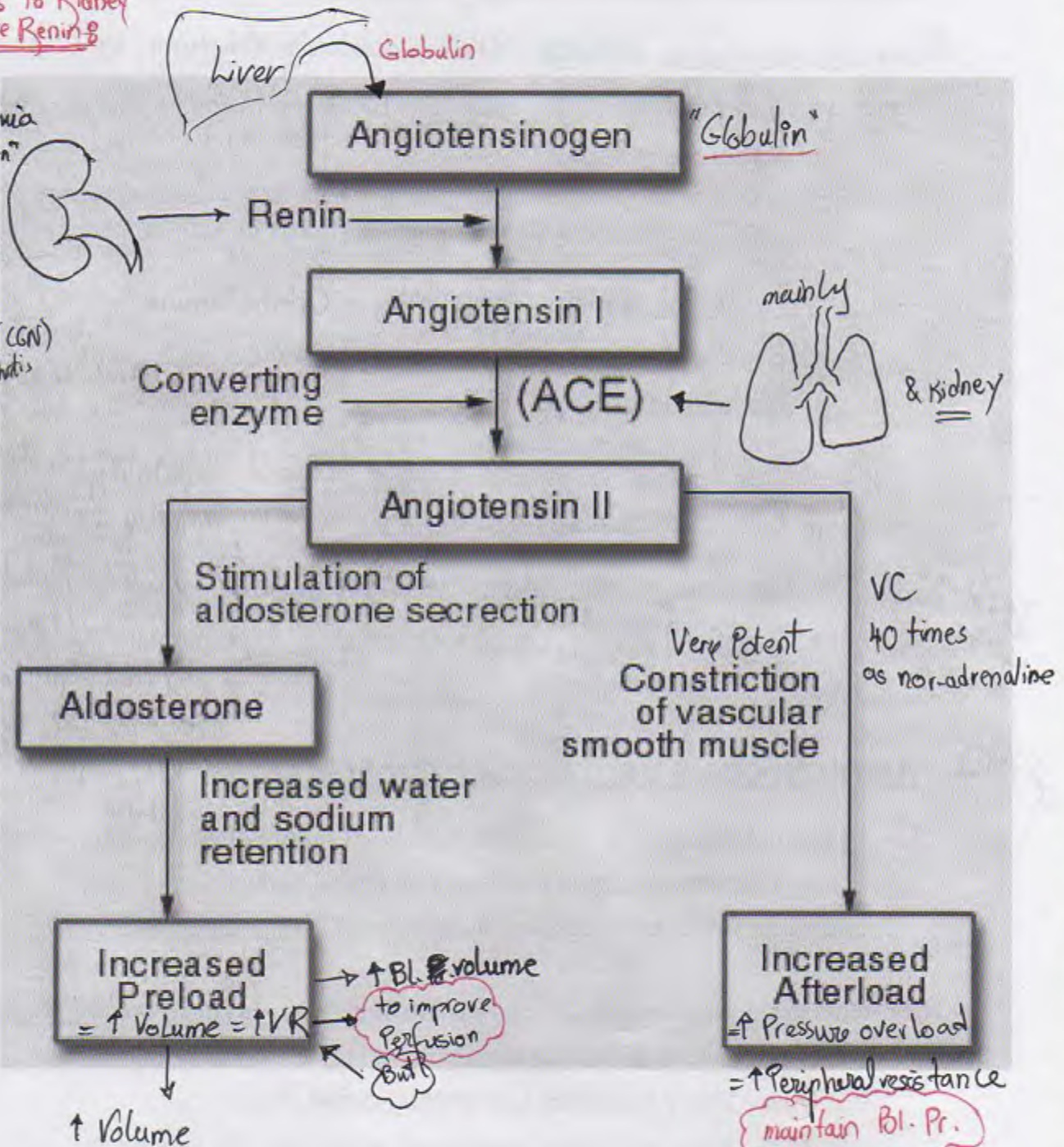
This Patient Needs CRT "Cardiac Resynchronization Therapy."

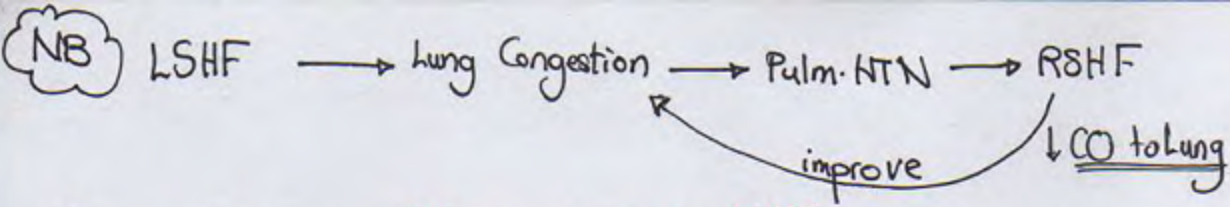
RAAS

Stimulants to Kidney to Secrete Renin

- ① Quantity Renal ischemia "↓ bl. perfusion"
- ② Quality Renal hypoxia
- ③ Renal damage (GN) Glomerulonephritis

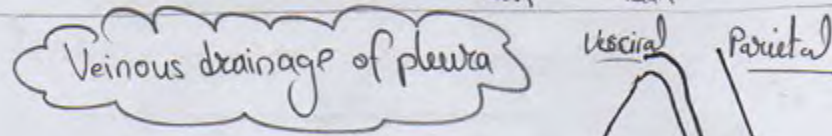
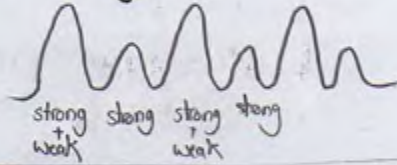
Zona Glomerulosa





Pulsus Alternans : Pathognomonic to LVF

- occurs in normal rhythm "Not in arrhythmia."
- Due to difference of Refractory period between strong & weak fibers in the diseased Myocardium. (weak fibers : longer refractory period)
- Strong fibers ~~beat~~ contract every beat.
- weak fibers contract every other beat



in Lt side HF
there's pleural effusion
"transudate"
due to back Pr. from Lt. Vent.
from Visceral pleura



Lt Ventricle

as Lung

Pulmonary Vens

in Rt side HF
there's pleural effusion
"transudate"
from Parietal pleura due to
back Pr. from Rt Ventricle



Rt Ventricle

intercostal

Systemic Vens

as chest wall

CLINICAL PICTURE

I. LEFT - SIDED HEART FAILURE

① Symptoms

- 1) Ss of low CO: *forward failure.*
- 2) Ss of pulmonary congestion: *backward failure.*
- 3) When RVF occurs: *symptoms of pulmonary congestion improve.*

② Signs "Examination"

1) General: "Examination"

- this reflex normally absent* → *V/S*
- a) RESPIRATION: *RC ↑ ⊕* → *↑ Congestⁿ ↔ "stretch" receptors* → *↑ "Churchill cope reflex"* → *↑ ☆ >20* not RSHF
 - b) PULSE:
 - Tachycardia: *except in patients receiving digitalis or β-blockers.* AVN block
 - Small volume: *except in conditions of high CO.*
 - Pulsus alternans: ☆ *alternating strong & weak regular beats detected by palpating the pulse or using the sphygmomanometer.*
 - c) SKIN: Coldness of the skin & Peripheral cyanosis.
 - d) CHEST: Pleural effusion & Bilateral basal crepitations. ☆
(Transudate)

2) Cardiac:

- a) Precordial examination: (inspection, palpation, percussion)
 - Signs of LV enlargement:
- b) Auscultation:
 - *Over the mitral area:* *→ tachycardia*
 - *Pro-diastolic (mid diastolic) - Left ventricular gallop (S3): "é filling" due to flabby myocardium. vibration*
 - *Pansystolic murmur of functional MR: due to LV dilatation.*
 - *Over the pulmonary area:*
 - Signs of pulmonary hypertension: *→*

③ Manifestations of the cause

e.g. Typical chest pain in Myocardial infarction. CAD

II. RIGHT – SIDED HEART FAILURE

① Symptoms

- 1) Symptoms of low CO: (forward failure).
- 2) Symptoms of systemic congestion: (backward failure).

② Signs

1) General:

a) PULSE:

- o Tachycardia: except in patients receiving digitalis or B-blockers.
- o Small volume: except in conditions of high CO.

b) SKIN: Coldness of the skin & Peripheral cyanosis.

c) CHEST: Pleural effusion.

★ d) PROMINENT SIGNS OF SYSTEMIC CONGESTION:

congested non pulsating only in SVC obstruction (except in SVC obstruction)

- o Neck veins: Congested pulsating neck veins. ^{except in SVC obstruction}
- o Liver: Enlarged, tender, $\pm$ pulsating, $\pm$ Hepato-jugular reflux. ^{tricuspid}
- o Lower Limbs: Oedema of lower limbs & later on ascites.

★ e) CARDIAC CACHEXIA:

With severe chronic HF, cachexia occurs due to:

- o $\downarrow$ caloric intake: due to Anorexia, N, V & $\downarrow$ absorption (GIT congestion).
- o $\uparrow$ caloric loss: due to $\uparrow$ metabolic rate ($\uparrow$ O₂ needs of the failing heart).

2) Cardiac:

a) Precordial examination:

- o Signs of RV enlargement.

b) Auscultation: (Over the tricuspid area):

- o Right ventricular gallop (S₃): due to flabby myocardium.
- o Pancystolic murmur of functional TR: due to RV dilatation.

③ Manifestations of the cause

e.g. Chronic cough, dyspnea & wheezes in COPD.

NB Causes in Enlarged Tender Liver :-

- ① inflammatory : Hepatitis
- ② Neoplastic : Try , 2ry ^{الأنسجة}
- ③ Congestion : eg. Portal HTN , Heart failure
- ± ④ Fatty Liver (due to associated inflammation; NASH)

NB Edema ^{قبل} Ascites $\Rightarrow$ HF (Except if precoc)
 Ascites ^{قبل} Edema of ~~Liver~~ $\Rightarrow$ Portal HTN

NB Pulsating Liver :- occurs in RSHF in severe late cases when Tricuspid regurg ~~occurs~~ (TR) occurs

Functional TR

RSHF $\leftarrow$ Systemic Congestion
 $\leftarrow$ Cardiac Cachexia

Symptoms Low CO + syst. Congestion

Signs

① General :

- ① Pulse * Tachycardia (X digitalis, X β -blocker)
 * Small Volume (X of high CO)

② Resp. usually not affected

③ Skin : Coldness, peripheral cyanosis

④ Chest : Pleural effusion

⑤ Systemic Congestion:

- * Neck veins : Congested pulsating
- * Liver : enlarged, tender $\pm$ pulsating $\pm$ hepatogastric reflux
- * L.L : edema * Late ascitis

⑥ Cardiac Cachexia : $\pm$ severe chronic HF

$\downarrow$ intake : anorexia, vomiting $\rightarrow$ Gut edema
 $\downarrow$ absorption $\rightarrow$ intestinal edema

↑MR : $\uparrow$ O₂ to failing heart

② Cardiac :

* Rt Vent. enlarged

① * auscult : - Rt : Vent. gallop (S₃)

- Pansyst. murmur of functional TR

Cause Manifest : COPD (cough, dyspnea, wheeze)

LSHF $\leftarrow$ Tachypnea $\rightarrow$ Pulm. Cong.
 $\leftarrow$ Pulsus alternans
 $\leftarrow$ Bilateral Basal Crepitations $\rightarrow$ Pulm. Cong.

Symptoms Low CO + Pulmonary Congestion
 when LSHF $\rightarrow$ RSHF $\Rightarrow$ Pulm. Congest² improves

Signs

① General :

- ① Pulse : * tachycardia (X digitalis, X β -blocker)
 * Small Volume (X high CO)
 * Pulsus alternans (X in arrhythmia)

② Resp : tachypnea (pulmonary congestion)

③ Skin : Same

④ Chest : same [not same etiology]

② Cardiac : - * Lt Vent. enlarged.

① auscult : Lt Vent. galloping S₃

- Pansyst. murmur : functional MR.

② P $\rightarrow$ Pulm. HTN : accentuated ~~2nd~~ pulm Comp. of S₂

Cause Manifest : M.I. (typical chest pain)

* Biventricular failure:

* LVF Causes → RVF

- * Both together : - Myocarditis, Cardiomyopathy
- Hyperdynamic Circ. (thyrotoxicosis, Anemia)
- Disease ~~(~~of~~)~~ Pericardial

* Acute MR : AMI → Rupture of papillary Ms due to ischemia → Acute MR.

⊙ SBE → vegetations on (M) that are perforated → acute MR.
"infected thrombus"

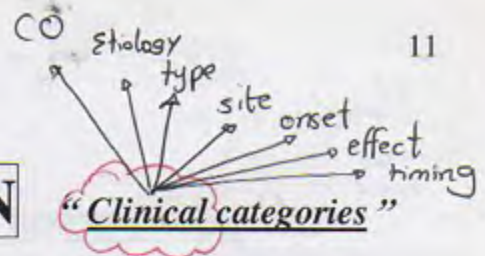
Signs

① General: (X of high CO)
② JVP: x of high CO
③ RFP: tachycardia (x of high CO)
④ Chest: rales (x of high CO)
⑤ S3: common (x of high CO)
⑥ S4: common (x of high CO)
⑦ Crackles: common (x of high CO)
⑧ Dyspnea: common (x of high CO)
⑨ Orthopnea: common (x of high CO)
⑩ Nocturnal cough: common (x of high CO)

Signs

① General: (X of high CO)
② JVP: x of high CO
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⑦ Crackles: common (x of high CO)
⑧ Dyspnea: common (x of high CO)
⑨ Orthopnea: common (x of high CO)
⑩ Nocturnal cough: common (x of high CO)

CLINICAL CLASSIFICATION



Site

1. LVF versus RVF.

(also Biventricular failure).

Onset

2. Acute versus chronic HF:

- Most causes produce chronic HF:

- HTN "need long time"
- Valvular "commonly rheumatic" "occur only in chronic" "within hours"

Maintained BP (compensatory mechanisms)
LL oedema
Ventricular enlargement

- Some causes produce acute HF as:

- LVF: may
AMI, Acute MR (after AMI or SBE).
- RVF:
Acute pulmonary embolism.
- Biventricular failure:
Acute myocarditis. (Global disease)

مقارنه شغوي

↓ BP
No LL oedema
No ventricular enlargement

Effect

3. Forward versus backward heart failure.

a) Forward failure:

- There are: manifestations of Low CO.
- Response to ttt: positive inotropics, arteriodilators.

b) Backward failure:

- There are: manifestations of congestion.
- Response to ttt: diuretics, venodilators.

so
ACE-I
are
1st
choice
in ttt

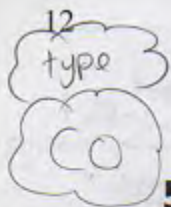
Timing

4. Systolic versus diastolic failure:

a) Systolic Failure: ↓ cardiac contractility, e.g. AMI, DCM.

b) Diastolic Failure: ↓ cardiac filling, e.g. CP, RCM, systemic HTN.

e.g. in Concentric Hypertrophy in HTN AS



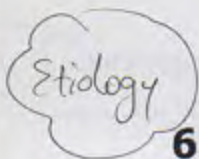
5. Low versus high CO failure:

a) Low CO failure:

- There is reduction of CO below normal.
- This type occurs in MOST CASES of HF.

b) High CO failure: *needs of tissues are higher due to* $\begin{matrix} \nearrow \uparrow \text{BMR} \text{ \& Hypoxia} \\ \searrow \downarrow \text{T.T.} \end{matrix}$

- There is usually peripheral VD & $\downarrow$ peripheral resistance, leading to $\uparrow$ VR & ultimately $\uparrow$ CO.
- There is $\uparrow$ CO, however, it is insufficient for the metabolic needs of tissues.
- This type occurs in cases of HYPERDYNAMIC CIRCULATION:
 - o Anemia, AR, Arteriovenous fistula.
 - o Beriberi, PDA, Paget's disease.
 - o Thyrotoxicosis, Liver failure.



6. Etiological classification:

"see before"

Etiology من الأسباب المرضية

FUNCTIONAL CLASSIFICATION



- A classification was developed by the New York Heart Association (NYHA) to assess the SEVERITY of heart failure:

- ie no symptoms e.g dyspnea*
- 1* CLASS I: No limitation of physical activity on ordinary effort.
- CLASS II: Slight limitation of physical activity on ordinary effort.
- CLASS III: Marked limitation of physical activity on less than ordinary effort.
- 2* CLASS IV: Symptoms occur even at rest. *few steps*

Hyperdynamic Circulation

Peripheral VD

Arteriolar VD $\rightarrow$ $\downarrow$ PR

$\uparrow$ CO

$\uparrow$ Velocity

$\uparrow$ T.T. transient time

Mechanism

$\downarrow$ Peripheral Resistance $\rightarrow$ $\uparrow$ VR
 $\rightarrow$ $\downarrow$ Diastolic B.I. Pr.

$\uparrow$ SV $\rightarrow$ $\uparrow$ CO $\leftarrow$ HR
 $\leftarrow$ Contractility
 $\rightarrow$ $\uparrow$ Systolic

Time that blood spend in the tissues for exchange (gases, waste, nutrient)

$\frac{\uparrow S}{\downarrow D}$ eg $\frac{170}{60}$!! $\rightarrow$ $\uparrow$ Pulse pressure $\rightarrow$ Big Pulse Volume (water Hammer Pulse)

Due to: ① $\uparrow$ VD $\rightarrow$ $\uparrow$ VR

② $\uparrow$ CO
 Marked in: Anemia, Thyrotoxicosis

Anemia

Due to Hypoxia $\rightarrow$ Anaerobic oxidation
 $\rightarrow$ Lactate & Pyruvate accumulation $\rightarrow$ VD

$\uparrow$ VR $\rightarrow$ $\uparrow$ CO
 $\uparrow$
 $\downarrow$ Viscosity

due to ① $\downarrow$ Hb $\downarrow$ viscosity

② $\uparrow$ VD $\rightarrow$ $\uparrow$ VR

③ $\uparrow$ CO

Thyrotoxicosis

Thyroxin $\rightarrow$ $\uparrow$ MBMR $\rightarrow$ more metabolites
 $\rightarrow$ lactate & pyruvate accumulation $\rightarrow$ VD

Thyroxin is +ve ino +ve chrono

$\uparrow$ VD $\rightarrow$ $\uparrow$ VR $\rightarrow$ $\uparrow$ CO
 Rapid Circ.

Liver Cell failure

① Liver Can't metabolize $\rightarrow$ metabolites accumulation
 $\rightarrow$ toxic subs. $\rightarrow$ VD

② Liver Cell failure $\rightarrow$ tissue injury $\rightarrow$ NO
 $\rightarrow$ adenosin } VD
 $\rightarrow$ TNF- α

$\uparrow$ VR $\rightarrow$ $\uparrow$ SV
 "Stroke Volume"

$\uparrow$ VD
 $\uparrow$ VR $\rightarrow$ $\uparrow$ CO
 Rapid Circ.

A-V fistula:

$\rightarrow$ Big enough (PDA)
 ①
 $\rightarrow$ Multiple

Short Circulation $\rightarrow$ bypass resistance $\rightarrow$ faster

$\uparrow$ VR $\rightarrow$ $\uparrow$ SV

less amount of bl. to tissue
 $\rightarrow$ pass faster

Etiology: ① Congenital: Multiple Congenital Vascular Anomalies
 -PDA

② Acquired: Traumatic

-iatrogenic

$\rightarrow$ accidental $\rightarrow$ uric acid
 $\rightarrow$ intentional (to perform Repeated dialysis in Renal failure)

Paget's disease

of Bone:

Bone destruction & Reformation

① Idiopathic activation of osteoclasts
 $\rightarrow$ generalized Bone destruction
 $\rightarrow$ Reformation by excessive fibrous tissue
 $\rightarrow$ Marked fibrovascular proliferation
 (Neovascularization: Angiogenesis)

Marked $\uparrow$ in Vascular Capacity $\rightarrow$ $\downarrow$ Peripheral R.

② Marked Bone destruction $\rightarrow$ IL-6 $\rightarrow$ Potent VD

$\uparrow$ VR $\rightarrow$ $\uparrow$ SV

$\uparrow$ VR
 $\uparrow$ CO

NB

Cytokines are released in

- ① Inflammation
- ② Infections
- ③ Immunological
- ④ Injury

Beri Beri:

① $\downarrow$ Vit B₁ (thiamine) $\rightarrow$ X CHO metabolism
 $\rightarrow$ pyruvate acc. decarboxylation $\rightarrow$ Potent VD

② B₁ is important for integrity of Capillary wall
 $\rightarrow$ if $\downarrow$ B₁ will lead to $\downarrow$ PR.

VR $\rightarrow$ $\uparrow$ SV

$\uparrow$ VR, $\uparrow$ CO

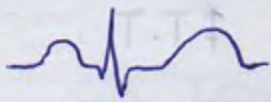
NB

- B₂ = riboflavin
- B₃ = Nicotinic (Pellagra)
- B₅ = Pantothenic acid (acne, parasthesia)
- B₆ = Pyridoxine (peripheral neuritis)
- B₇ = Biotin (6th retardation in infants)
- B₉ = Folic acid
- B₁₂ = Cyanocobalamin

? Megaloblastic anemia

White Knight Love

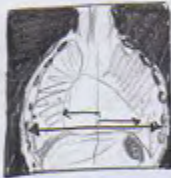
Investigations for HF



ECG

- Chamber enlargement
- Cause of HF
↳ CAD, BBB
- PPT: arrhythmia
AMI

CxR



- chamber enlarge.

- Pleural effusion
- Pulm. Congestⁿ (LHNF)

Imaging

Echo



chamber enlarge.

- Cause: (Valvular)

- Contractility
SF, EF

angiograph



- chamber enlarge.

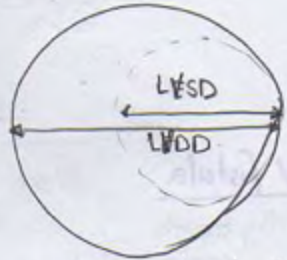
- Cause (CAD)

others



- Cause:
 - T₃, T₄
 - Cardiac enz.
 - CBC

Lt Vent End systolic diameter



Lt Vent End diastolic Diameter

لنرمز تعرف

NB HF is diagnosed Clinically

& investigations takes minor Role in Diagnosis

INVESTIGATIONS

I. ECG:

- Chamber enlargement.
- Cause of HF may appear e.g. CAD, BBB. → dilated Cardiomyopathy
- * - Precipitating factor may appear e.g. arrhythmias.

ECG - انما ما نستخدمه بيلا

II. IMAGING:

1. CXR:

- Chamber enlargement.
- * - Pulmonary congestion in left-sided HF, or Pleural effusion. ^{LVF RVF}



2. Echocardiography:

- Chamber enlargement.
- Cause of HF may appear e.g. valvular lesions.



CONTRACTILITY STATE OF THE MYOCARDIUM:

- SHORTENING FRACTION (SF) = $\frac{LVDD - LVSD}{LVDD}$ (N = 25-40%)
- EJECTION FRACTION (EF) = $\frac{LVDD - LVSV}{LVDD}$ (N = 60-70%)

3. Cardiac catheterization & angiocardiology:

- Chamber enlargement.
- Cause of HF may appear e.g. CAD.

to Reach Rt Vent (femoral V.)
to reach Lt Vent. or Coronaries (femoral A.)

متعطل قسطرة على طول
من صريح: يمكن السحب
يعرف هكذا حاصره ثاميه

III. Other investigations for the cause & ppt factor:

- ① - Cardiac enzymes: "CKMP" for myocardial infarction.
- ② - CBC: ↓Hb for anemia.
- ③ - Thyroid functions: ↑T₃, T₄ for thyrotoxicosis.

Just
Examples

TREATMENT OF HEART FAILURE

GUIDELINES for TTT (PLAN of TTT)

1. Treatment of the underlying etiology..... e.g...
2. Treatment of the precipitating factors..... e.g...

3. Treatment specific to the syndrome of HF:

▪ Decreasing the cardiac load:

- a) Rest: Physical & emotional.
- b) Reduction of preload:
 - o Diet control, especially salt restriction.
 - o Diuretics.
 - o Vasodilators (Venous).
- c) Reduction of afterload:
 - o Vasodilators (Arterial).

ACE-I or ARB

▪ Decreasing the myocardial damage:

- β -blockers. → in chronic

Never in acute

▪ Increasing the myocardial contractility:

- a) Digitalis.
- b) Other inotropic agents:
 - o Sympathomimetic amines: dopamine & dobutamine.
 - o Phosphodiesterase inhibitors: amrinone & milrinone.

▪ Resynchronizing the ventricles:

"CRT"

- In patients with cardiomyopathy who have the problem of "intraventricular conduction delay" or bundle branch block.

▪ Symptomatic treatment:

- a) For hypoxemia: oxygen administration.
- b) For cardiac dyspnea: aminophylline administration.

severe pulmonary congestion → bronchospasm !!

4. Treatment of refractory HF. → not of PPT factors

5. Treatment of acute pulmonary oedema.

in acute LVF

Emergency

راحة بالفراش مع مراعاة تعليمات المريض
 "لا يتم سلق في الروش"

PHYSICAL REST

- **BENEFITS:** Bed rest reduces the metabolic needs of the body & the cardiac load.
- **POSITION:** Semisitting position is preferred to decrease the venous return.
 - better than flat Position to improve efficacy of diaphragm & intercostal Ms.
- **COMPLICATIONS** of prolonged bed rest:
 - Venous stasis → DVT → & PULMONARY EMBOLISM. ~~stagnation~~
 - Pneumonia. → orthostatic Pneumonia "positional" due to stagnation of secretions. (by bact. infection)
 - Constipation & retention of urine. "atony"
 - Muscle wasting & osteoporosis. (i.e. bone wasting) "disuse atrophy"
 - Bed sores.
 - Psychoneurosis.

EMOTIONAL REST

"not addictive
 But S.E. is tolerance"

- **SEDATION** may be achieved by: Diazepam 2 – 5 mg tds orally.

DIET

- **Salt restriction:** decreases blood volume & thus decreases the preload.
- **Fluid restriction:** only needed in severe cases of HF.
- **Small frequent meals:** to decrease the work of the heart.
- **Reduction of body weight in obese patients.** (fat tissues have large Bl supply)

VASODILATORS

"Most important Drugs"
 "No addiction to these drugs"
 tolerance ~~small~~

Action

1. Reduction of preload by: veno – dilatation.
2. Reduction of afterload by: arteriolar dilatation.

Types

VASODILATOR	Site of action	Mode of administration
Nitrates (Isosorbide dinitrate)	Venous	20 – 40 mg / 8h orally
Hydralazine	Arterial	50 mg / 6h orally
Na nitroprusside	Arterial & venous	0.5 – 10 µg / kg / min, IV infusion
Prazosin (α – blocker)	Arterial & venous	orally
ACE inhibitors: Short acting: Captopril Long acting: Ramipril	Arterial & venous	6.25 – 25 mg / 8h orally 1.25 – 2.5 mg / day orally
ARBs: Candesartan	Arterial & venous	Initially 4, target 32 mg / day orally

important
 not most
 very potent

sublingual

Important points

- In chronic HF, ACE-I is the most useful, esp. in LV systolic failure. ??

- Contraindications to ACE-I in HF:

- Can give ARB }
"same effect" }
1. Intolerance due to the presence of side effects, e.g. dry cough.
2. HYPERKALEMIA: serum potassium > than 5.5 mEq/liter.
3. Hypotension: SBP < 90 mm Hg.
4. Bilateral renal artery stenosis: because they will cause renal failure.
5. Caution in renal failure with serum creatinine > 3.0 mg/dL & stop the drug if: serum creatinine rises by more than 30 % of the base-line level.

- When ACE-I is contraindicated in HF, alternative good VD's are:

1. Combination of Nitrates (venodilator) & Hydralazine (arterio dilator).
2. ARBs: especially Candesartan, (in case of intolerance to ACE-I).

DIURETICS

- They are very useful in the treatment of HF.
- They promote loss of salt & water, thus: ↓ blood volume & ↓ preload.

- Types:

1. Thiazides.
3. Potassium sparing diuretics.

2. Loop diuretics. ^{الأقوى}
4. Others diuretics.

1. Thiazides

- Action: on the distal tubules, to ↓ reabsorption of: Na, H₂O, K, Cl, Mg ^{مزره}

- Preparations:

- Hydrochlorothiazide: 50 – 100 mg/day.
- Chlorothalidone: 50 – 100 mg/day.

- Side effects:

- "Give K⁺ oral supplement"
1. Hypo kalemia: which may precipitate digitalis toxicity.
 2. Hypo chloremic alkalosis: which may lead to tetany.
 3. Hypo natremia.
 4. Hypo magnesemia.
 5. Hyper uricemia. (↓ U.A. excretion: Competition)
 6. Hyper glycemia. ↓K → ↓β cells → ↓insuline
 7. Hyper lipidemia. (Thiazide ⊖ lipoprotein lipase)
 8. Hyper calcemia.
- HCO₃⁻ & Cl⁻

Q1) why ACE-I used in chronic? أقراص مضيق شريان الأبهر

① why drycough? side effect of ACE-I as it $\ominus$ Kininase $\rightarrow$ accumulation of Kinin $\downarrow$ dry irritating cough

② ACE-I $\rightarrow \ominus K^+$ secretion by Renal tubule $\rightarrow$ Hyperkalemia.

③ ACE-I administration $\rightarrow$ Monitor Renal Function "Creatinine < 1.3 mg/dl" high $\uparrow$ if More than 1.3: Renal impairment if > 3.0 Renal failure

Must stop ^{due to} * deteriorated GFR by the drug
* Hyperkalemia is exaggerated $\leftarrow$ effect of drug by renal failure

Bilateral

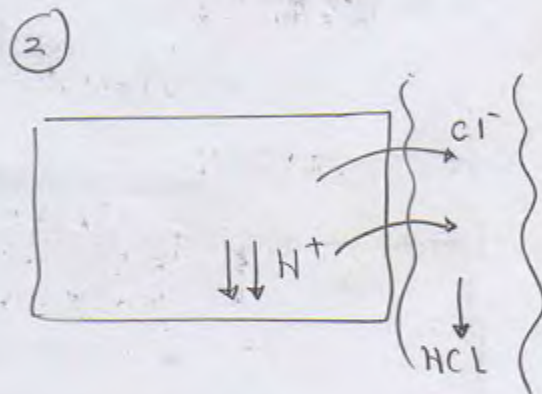
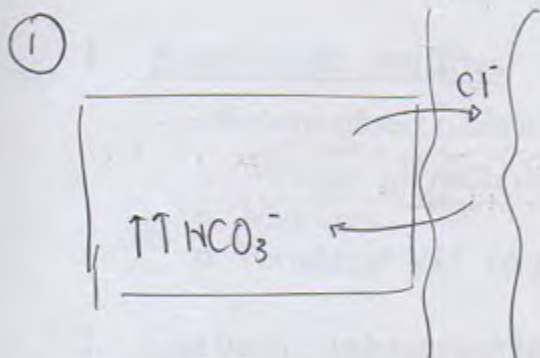
④ Renal Artery stenosis:

as ~~there's~~ $\downarrow$ GFR: Angiotensin II $\rightarrow$ VC selectively in Efferent More than aff. this $\downarrow$ normally occurs in Renal Artery stenosis

when Give ACE-I $\rightarrow$ $\times$ synthesis of Angiotensin II $\rightarrow$ $\downarrow$ GFR $\rightarrow$ Renal failure

Hypochloremic Alkalosis

2 mechanisms in different Parts in the tubules:



2. Loop diuretics

- **Action:** on the ascending limb of loop of Henle, to ↓ reabsorption of: Na, H₂O, K, Cl.
THEY ARE THE MOST POTENT DIURETICS.
- **Preparations:**
 - Frusemide (Lasix): 40 – 200 mg/day (orally or IV).
 - Ethacrynic acid: 50 – 100 mg/day (orally or IV).
- **Side Effects:**
 - Same as Thiazides (but without hypercalcemia).

3. Potassium sparing diuretics

- **Action:** on the distal tubules to ↓ reabsorption of: Na, & H₂O,
BUT: they have the advantage of retaining K (they ↓ the secretion of K).
THEY ARE WEAK DIURETICS:
 - Used to potentiate the action of Thiazide or loop diuretics.
 - Used to avoid the potassium losing effect of Thiazide or loop diuretics.
- **Preparations:**
 - Spironolactone (aldosterone antagonist): 100 – 400 mg / day.
 - Triamterine.
 - Amelioride.
- **Side effects:**
 - Hyperkalemia & Metabolic acidosis. *association*
 - Gynecomastia with prolonged use. *"steroid ring"*

Aldosterone spironolactone
- ↑ Na, H₂O is Aldosterone
- ↓ K *cause* Antagonist
- ↓ H⁺ *cause* Hyperkalemia
Metabolic acidosis.

K⁺ "Hyperkalemia"
Metabolic acid
H⁺ retention (ald. antagonist)
extra cellular shift of H⁺
in exchange with K⁺

4. Other diuretics

1. Natriuretic peptides:

- Infusions of recombinant NP have been shown to cause:
 - Afferent arteriolar dilation & efferent arteriolar constriction → ↑ GFR.
 - Natriuresis.
 - Peripheral VD (A & V).

2. Carbonic anhydrase inhibitors:

- Diuretics: ↓ reabsorption of: NaHCO₃ in the proximal tubules.
- Also: correct the metabolic alkalosis that occur due to thiazide diuretics.

$\text{C} \rightarrow \frac{\text{safety}}{\text{efficacy}} > \text{الأمان والفعالية}$
 Choice 4

DIGITALIS

"Digitalis is no longer extensively used in the management of HF, although it is an effective positive inotropic agent."

ACTIONS

the ino

► It increases the myocardial contractility: through:

Competitive inhibition of sodium-potassium ATPase $\rightarrow$ $\uparrow$ intracellular Na. Then, Na-Ca exchange occurs & this increases the intracellular Ca. High intracellular Ca promotes sliding of actin & myosin.

This leads to increased force of myocardial contractility; which causes:

- Increased cardiac output.
- Decreased venous congestion.
- Decreased cardiac size.

vechono

► It slows the heart rate: through:

- Vagal stimulation.
- Direct inhibition of the SAN.

معدل

► It increases the excitability of the atria & ventricles:

- In digitalis toxicity, arrhythmias may occur.

► It inhibits the conduction of the AVN:

- In digitalis toxicity, AV block may occur.
- Digitalis can be used to protect the ventricle in atrial arrhythmias. $\left. \begin{array}{l} \text{600 bpm} \\ \text{AV block} \end{array} \right\} \text{vent: } 120$

► Diuretic effect:

- Increased renal blood flow.

► On ECG:

- Digitalis effect: Sagging depression of ST segment, Flat or inverted T wave.
- Digitalis toxicity: Different types of arrhythmias.

INDICATIONS

1. Heart Failure.
2. Rapid atrial arrhythmias: (A-V nodal block) " $\downarrow$ Conductivity "
 - Atrial fibrillation (AF).
 - Atrial flutter.
 - Supraventricular tachycardia.

يا ساروم لو الاسين مع بعض

Highly indicated

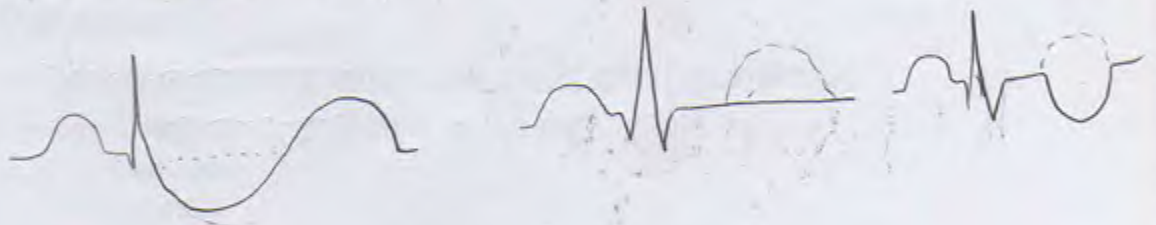
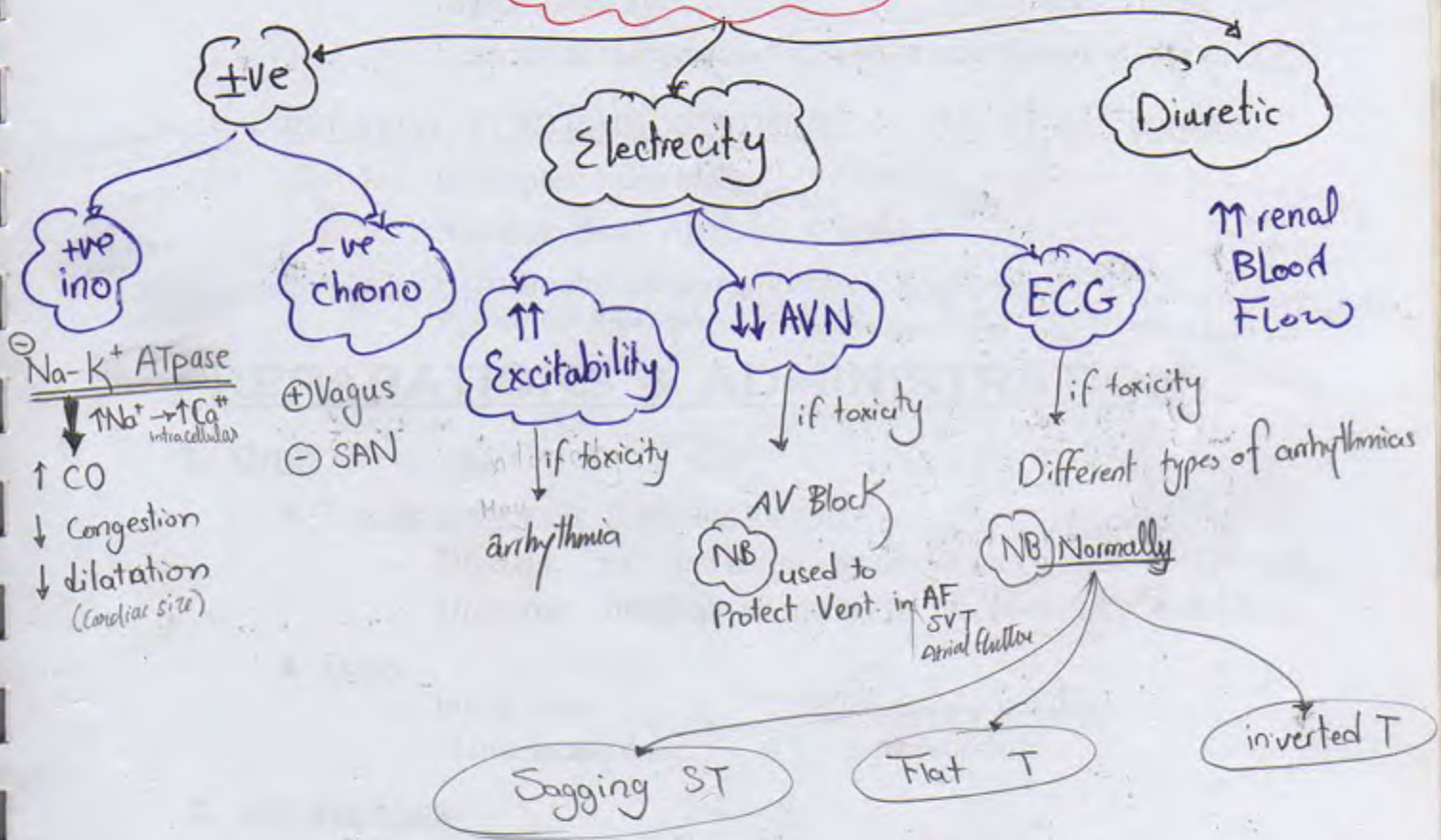
الأمان والفعالية

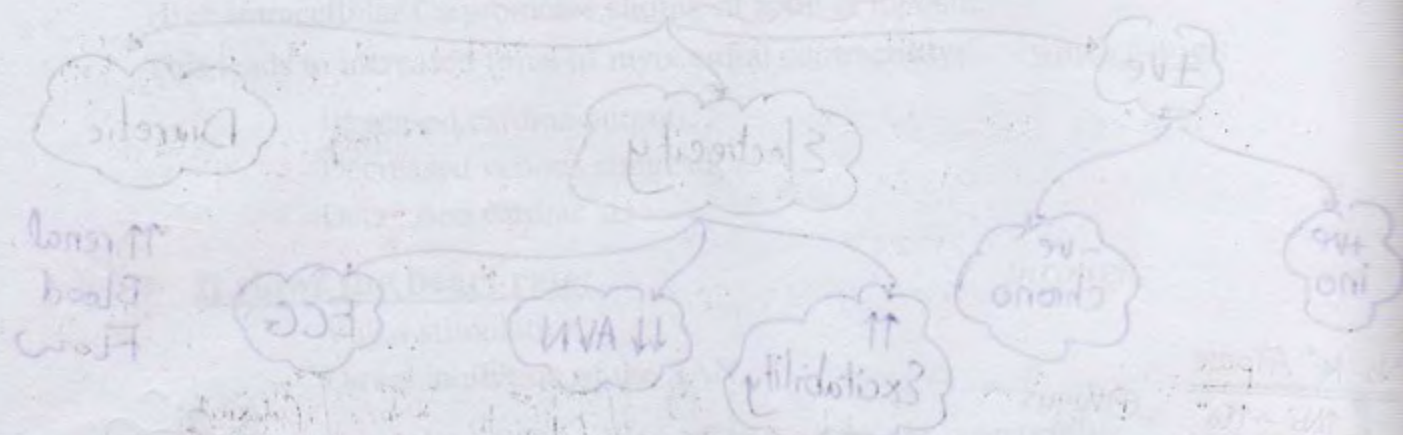
* Digitalis & Sympathomimetics $\rightarrow$ Arrhythmia !!
 ↓ in HF ↓ in Bronchial Asthma
جیو پی جی وی

* Never Give Digitalis in Ventricular tachycardia as It will convert VT
 "Vent. tachy" 200 to VF (fibrillation) 600

↳ Digitalis is Not Anti arrhythmic But is Given in Some Cases of arrhythmia.

Digitalis Actions





* Very Slowly LV:

Aminophylline : arrhythmia

β - stimulant

Ca^{+} : Systole

Digitalis :

Digitalis is highly indicated in:

- ① Patients with HF & rapid atrial arrhythmias, esp. AF.
- ② Patients with HF not responding to: Vasodilators, BB & Diuretics.

CONTRAINDICATIONS

• ABSOLUTE CONTRAINDICATIONS:

- Digitalis toxicity.
- Ventricular tachycardia (Digitalis may change it to fatal VF). *Lidocaine or DC shock* *narcotic*

• RELATIVE CONTRAINDICATIONS:

"It is better avoided:"

⊖ AVN - Incomplete heart block.

⊖ AVN - Nodal rhythm. (digitalis ~~SAN~~ *Purkinje*)

- Hypertrophic cardiomyopathy. *Affect only Lt Vent.*
asymmetrical Hypertrophy (septum) free wall → *resembl AS* → *↑ contraction ⇒ ↑ AS* ⇒ *digitalis*

PREPARATIONS & ADMINISTRATION

1. Oral:

▲ It is the usual route of administration:

- Digoxin: excreted mainly by the *kidney* (tab = 0.25 mg).
- Digitoxin: metabolized mainly by the *liver* (tab = 0.1 mg).

▲ Dose:

- Initial dose: 2 tablets daily for 5 days.
- Maintenance dose: 0.5 – 2 tablets daily.

2. Intravenous:

▲ Indicated in:

Acute LVF
 "Acute pulmonary oedema & rapid atrial arrhythmias".

- Digoxin (amp. = 0.5 mg). *Not digitoxin*
- Cedilanid.
- Ouabain.

▲ Dose:

- 0.5 – 1 mg in 5% glucose over 30 min, *very slowly IV* THEN:
- 0.5 mg / 6 h till a therapeutic response appears, THEN:
- Continue with oral digitalis.



DIGITALIS TOXICITY

Overdose
Hypokalemia
Renal, hepatic impairment

1. Clinical Picture:



▲ Gastrointestinal:

- Anorexia: usually the earliest first symptom.
- Nausea & vomiting.



▲ Cardiovascular: "Different types of arrhythmias & heart block"

- Premature beats, especially occurring in *bigemini* or *trigemini*. *extrasystole*
- Paroxysmal atrial tachycardia: *with heart block.*
- Paroxysmal ventricular tachycardia: *most serious.* *in attacks* *→ VF anytime!*

Any type of arrhythmia
But these are most common

▲ Neurological:

- MENTAL disturbances, e.g. psychosis
- PAIN: headache & neuralgias.
- VISION: **Blurring** of vision & **coloured** vision (yellow or green).

Peripheral Ns
→ Pain
CNS → Psychosis
Headache

▲ Miscellaneous:

- Increased blood coagulability. [*abnormal activation of the clotting system*]
- Gynecomastia, with prolonged use.



2. Treatment:

- ① Stop digitals.
- ② Correct hypokalemia, if present:
 - Stop drugs causing hypokalemia e.g. loop diuretics.
 - Give potassium: Orally or IV.
- ③ Digitalis antibodies.

④ TTT of the manifestations of digitalis toxicity:

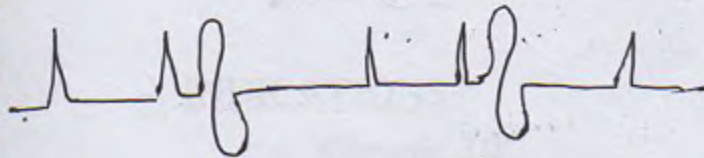
- For vomiting: Anti-emetic drugs, e.g. metoclopramide.
- For arrhythmias:
 1. Anti-arrhythmic Drugs: especially epanutin & lidocaine. *phenytoin* *Xilocain*
 2. Atropine: for heart block & bradycardia. *class 1*
 3. DC: Avoided because it may induce more serious arrhythmias. Allowed ONLY in case of the fatal arrhythmia: VF.



Sinus



Single extrasystole = Premature Beat



Trigemini



Bigemini

Psychosis: Severe Mental disturbance in, as the Pt Can't differentiate ^{Frontal} what is real from what is imaginary.

① Digitalis

② Corticosteroid

③ Anti tuberculous: INH



DC shock Can be used in any type of arrhythmia by any cause except Digitalis otherwise if used in Digitalis toxicity → More serious arrhythmia

So Never Used in Digitalis toxicity - except in fatal arrhythmia: VF

Important

BETA - BLOCKERS

New

- تعالج في المستشفى
- In the past: they were absolutely contraindicated in HF due to their -ve inotropic effect.
 - Recently: they are used to treat HF because they were found to:
 - o Decrease the direct myocardial damage induced by catecholamines in HF.
 - o Improve the prognosis.

- INDICATIONS:

- o Chronic HF.
- o Moderate HF (NYHA classes II & III).

- CONTRA - INDICATIONS:

- منه اي حال
- o Acute HF (e.g. shortly after AMI).
 - o Severe HF (NYHA class IV).

لا علاج في AMI من غير HF فان نيكو β -B
منه لعل acute HF
Never !!! β -blocker

- ADMINISTRATION:o TYPES:

صن اي β -blocker

Only: **Second generation** β_1 blockers (e.g. Metoprolol), or:
Third generation β_1 blockers (e.g. Carvedilol).

Proprenolol
Atenolol
★ No Benefit of 1st Gen.

o DOSE: "Gradually increasing doses" ★

- نفس طريقة ARBs
P13
صن اي dose
- We start with extremely low doses (one-eighth of the target dose).
 - We gradually $\uparrow$ the dose every 1 – 2 weeks to reach the target dose.
 - Target dose: 25 – 50 mg of carvedilol daily.

★ Combined β & α blocker

Not responding to classic ttt
for few days

Treatment of Refractory Heart Failure

Etiology of refractory HF:

Mech

1. Presence of a mechanical factor hindering the cardiac function: e.g.
 b.g. • Severe valvular disease.
 o.g. • Pericardial effusion or constrictive pericarditis.

Cause

2. Persistence of the cause: e.g. Uncontrolled Hypertension.
3. Presence of a precipitating factor: e.g. Chest infection.
 Arrhythmia
4. Improper management: e.g.
 • Inadequate rest.
 • Inadequate salt restriction.
 • Inadequate digitalis.

PR

5. Terminal cases of HF: with advanced myocardial damage,
 e.g. Massive myocardial infarction.
 DCM

Treatment:

1. Removal of the mechanical factor: e.g. surgical ttt of valvular disease.
2. TTT of the cause: e.g. proper control of Hypertension.
3. Removal of the precipitating factor: e.g. proper antibiotics for chest infection.
 Antiarrhythmics for Arrhythmia
4. Proper management:
 • Adequate rest.
 • Adequate salt restriction ($< 1\text{gm} / \text{day}$) & fluid restriction in severe cases.
 • Adequate digitalis.
5. For Terminal cases of HF: Aggressive ttt.
 a) Diuretics: use combinations e.g. loop diuretic & potassium-sparing diuretic.
 b) Vasodilators: use combinations of IV vasodilators such as Na nitroprusside and a potent IV inotrope such as dopamine or dobutamine.
 c) Mechanical measures: (ventilation)
 • Mechanical removal of fluid using phlebotomy or dialysis.
 • Mechanical assistance of circulation using intra-aortic balloon counterpulsation:
 "Balloon is inflated in diastole & deflated in systole". V. invasive
 d) Cardiac transplantation: in patients below the age of 60.

symptomatic
لغالب
Can't give for NYHA IV

ICU

Computerized

المرضى في المستشفى

Treatment of Acute Pulmonary Oedema

- Oxygen administration
- Morphine IV
- Furosemide IV
- Vasodilators
- Diuretics
- Positive end expiratory pressure
- Extracorporeal membrane oxygenation

Respiratory distress syndrome

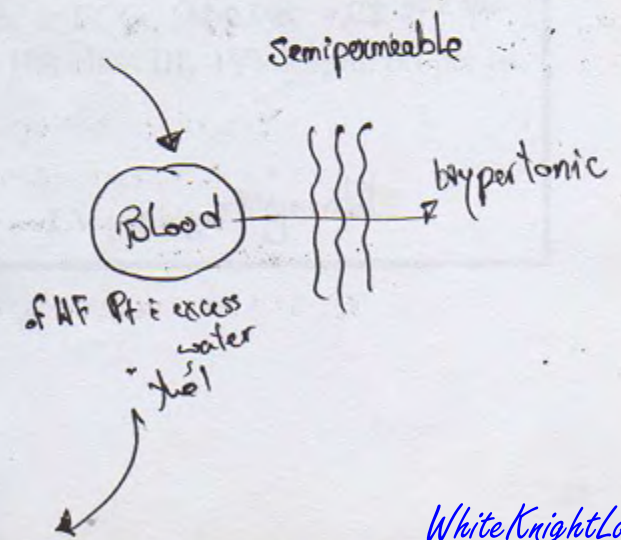
Indications

In patients with confirmed or highly suspected pulmonary oedema, if the patient is hypoxemic, tachypnoeic, and has a clear lung field on auscultation, the following treatment should be initiated:

Phlebotomy

- open & Removal of Blood from Ven
- closed : Rotating tourniquet

open & closed
Venesection



Emergency

Oral

Written

23
P-11
Morphine
Frusemide

Massive intra alveolar transudation $\rightarrow$ Hypoxia

Treatment of Acute Pulmonary Oedema

All IV

symp.
ORC

losix

1. Hospitalization in ICU: bed rest in ^{semi} sitting position.
2. Oxygen administration: 6 – 8 litres / min.

3. **Morphine IV 5 mg**, to relieve the anxiety and decrease the sympathetic stimulation, thus causing VD.

4. **Frusemide IV 40 mg**, to correct the hypervolemia, to be repeated every ½ hour until relief.

The 2 most important measures

dyspnea
Bubbling crepitations

5. Vasodilators IV:

AoV

Na nitroprusside:

IV infusion (0.5 – 10 μ g / kg / min).

- Nitroglycerine:

IV infusion.

6. Positive inotropics IV:

- Digitalis,

esp. in cases associated with AF.

- Dopamine or Dobutamine. "dose dependent"

no VC
true no VC

7. Aminophylline IV (250 – 500 mg), slowly, to relieve the bronchospasm.

8. Treatment of the cause & the precipitating factor. (Acute Lt Vent failure)

Antibiotics to chest infections

e.g AMI: streptokinase (reperfusion)

9. In refractory conditions:

Mechanical

Mechanical removal of fluid using phlebotomy or dialysis.

Mechanical assistance of circulation using intra-aortic balloon counterpulsation.

M.V.

Mechanical ventilation.

Resynchronizing the ventricles:

"CRT"

Indication

- In patients with cardiomyopathy who have the problem of:
"intraventricular conduction delay" or bundle branch block:
 - As evidenced by wide QRS ≥ 0.12 sec in ECG. (N=0.1 sec = 2 1/2 s. squares)
 - In patients with severe symptoms (NYHA class III, IV) despite proper ttt.

Method

- Biventricular pacing ^{intra Ventricular Conduction delay} or - LV pacing ^{segmental}

CRT = Cardiac Resynchronization Therapy

Clinical manifestations of Cardiac Disorders

Symptoms of cardiac disorders

1. PULMONARY CONGESTION SYMPTOMS:

- These occur in left sided heart failure due to failure of the heart to accept the pulmonary venous return:
 - Dyspnea.
 - Cough.
 - Hemoptysis.
 - Recurrent Chest infections.

2. SYSTEMIC CONGESTION SYMPTOMS:

- These occur in right sided heart failure due to failure of the heart to accept the systemic venous return:



edema - ascites

- Confusion & insomnia. (*steep Center in Hypothalamus*)
- Right hypochondrial pain.
- Anorexia, nausea, vomiting, dyspepsia & epigastric pain.
- Oedema of dependent parts (LLs or sacrum).
- Abdominal swelling due to ascites.

3. LOW CARDIAC OUTPUT SYMPTOMS:

- These occur in left or right sided heart failure or in both due to failure of the heart to eject a sufficient CO:
 - BRAIN: Dizziness, headache & syncope.
 - EYES: Blurring of vision.
 - HEART: Angina pectoris.
 - KIDNEYS: Oliguria.
 - MUSCLES: Easy fatiguability & Intermittent claudication. *Limping*
 - SKIN: Pallor & coldness; peripheral cyanosis in severe cases.

↓
Mild

↓
Sever

4. PALPITATION:

- This is an unpleasant awareness of the heartbeat.

- This occurs due to:

thyrotoxicosis
Most common cause of palpitation

- Change in rate:
- Change in rhythm:
- Change in force:
- Psychoneurosis.

e.g. *Tachycardia or Bradycardia.*
Arrhythmia.
Forceful cardiac contraction.
"Volume overload"

100/70 → 160

5. CHEST PAIN:

- Pain of cardiac origin may arise from:

- Myocardium: e.g. angina pectoris or AMI.
- Pericardium: e.g. pericarditis or pericardial effusion.
- Endocardium: e.g. mitral valve prolapse. *v. benign*
- Aorta: e.g. dissecting aortic aneurysm.
- Pulmonary: e.g. acute pulmonary embolism.

stretch of the Pericardium
Mediastinal irritation

↳ Pleurisy

6. PRESSURE SYMPTOMS:

- These occur due to pressure of enlarged cardiac chambers on the surrounding structures.

- An enlarged left atrium, aortic aneurysm or pericardial effusion may compress:

- Oesophagus: dysphagia.
- Tracheobronchial tree: dyspnea & cough.
- Left RLN: (*Aorta*) hoarseness of voice.
- Spine: back pain.
- SVC: swelling of the face & puffiness of eye lids.

Congested non-pulsating Neck Veins

7. CYANOSIS:

- Bluish discolouration of skin & may be mucous membranes due to the presence of more than 5 gm reduced (unoxxygenated) Hb / 100 cc of blood.

- *Fallot* - *Eisenmenger*

"Pulm. Congestion"
LVF

DYSPNEA

- It is a subjective distress associated with difficulty in breathing.

TYPES

1. Exertional:

5 grades (American thoracic society scale)

- Grade 0: none no dyspnea.
- Grade 1: mild dyspnea on marked exertion. (3 floors)
- Grade 2: moderate dyspnea on moderate exertion. (2 floors)
- Grade 3: severe dyspnea on mild exertion. (1 floor)
- Grade 4: very severe dyspnea on minimal exertion.

2. Dyspnea at rest. On no exertion

3. Orthopnea: Orthopnic decubitus.

- What happens ??

- o It is dyspnea on recumbency that is relieved by sitting up.

- Pathogenesis:

recumbency results in:

- o ↑ VR to the heart → more pulmonary congestion & more dyspnea.
- o Elevation of the diaphragm & improper use of chest wall muscles.
(Pr. by abd. Viscera)

4. Paroxysmal nocturnal dyspnea: PND

- What happens ??

Classic PND: ⇒ RD ^{stress} ⇒ ⊕ sympathetic ⇒ severe sweating

- The patient is aroused 1 – 2 hours after sleeping with:

- o Severe dyspnea, Sweating, Cyanosis, Cough,
- o Expectoration: frothy blood-tinged. (Hemoptes, 3) غلى ودماء

- The patient sits up in bed or stands beside a window to get more air.

Cardiac asthma: (asthma = bronchial obstruction leading to severe dyspnea)

- ⊕ Generalized wheezes: due to bronchial oedema & bronchospasm.

Acute Pulmonary oedema:

- ⊕ Generalized bubbling crepitations: due to intra-alveolar oedema.

Cyanosis

Coars
Lung
جفيرة

Acute LVF

& interstitial

Dyspnea

1. Exertional

- Grade 0 → No
- Grade 1 → Marked
- Grade 2 → Moderate
- " 3 → Mild
- " 4 → Minimal

2. at rest

decubitus

3. orthopnea

- inrecumbency
- relieved by sitting
- Cause
 - ① ↑TVR
 - ② elevation of diaphragm

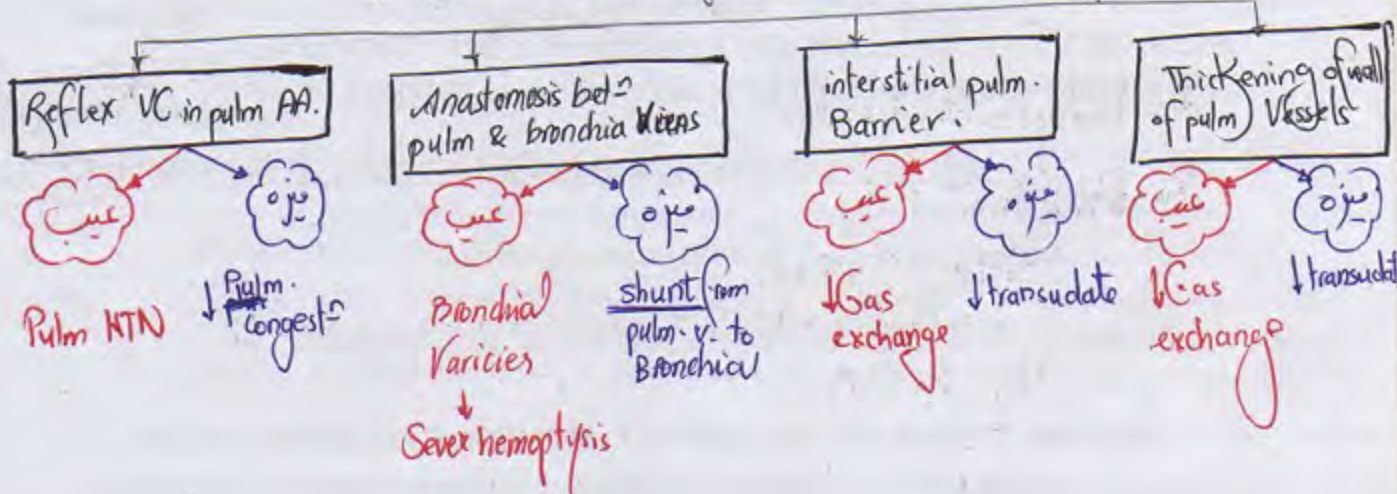
4. PND

due to Recumbency ↑ Vagal tone

- ① Classic aroused ~ 1-2 hrs after sleep
dyspnea, Sweating, Cough, Cyanosis
Expectoration of frothy Blood tinged sp.
- ② Cardiac asthma
⊕ Bronchospasm, bronchial edema
- ③ Acute pulm edema
⊕ Generalized bubbling crep. (intra alveolar edema)

acute process eg in AMI...

in Case of MS as it's gradual protective mechanisms occur



in Cardiac Asthma

↳ Bronchial edema ⊕ imitate Masquerade Vagal Receptors → Broncho spasm.
 ↳ ↑ Pulm edema.
 ↳ LVF

acute Pulm. edema

↳ Rapid accumulation of fluid in the interstitial fluid & inter alveolar space
 ↳ Diffuse alveolar & interalveolar

سعال

Sweaty Pale BradyCardia Hypotensive ⇒ VasoVagal attack
 "parasympathetic"

NB When MS cause **AND**? i.e. Acute Pulm. edema

- ~~the~~ acute Onset pathology on top of the chronic pathology of MS
e.g. ~~the~~ Atrial fibrillations → Rapid atrial bt. stagnation.

or MI

600 beat/min.

Absent a wave in CVP

Absent P wave in ECG

NB Normally Systemic Circulation > Pulm. Circulation

shunting → ~~test~~ (when ↑ Pulm. HTN) → to systemic V.

Example like Portal HTN → esophageal Varices

↑ its Pr. → Branchial
Varices
"dilated Branchial V."
مع الزرق، الدم من

Rupture

Sever hemoptesis

& die

- Pathogenesis:

- Recumbancy increases the VR to the heart → more pulmonary congestion.
- Decreased sympathetic activity during sleep → reduction of inotropism.

- Incidence:

- PND occurs more commonly in acute LHF as it is an acute process.
- PND occurs less commonly in MS because it is a gradual process allowing time for protective mechanisms to take place to ↓ further pulmonary congestion.

a. Reflex vasoconstriction of pulmonary arteries: (تقلص الشرايين الرئوية)

This will decrease the pulmonary congestion, but on the other hand it will lead to development of pulmonary hypertension. → RVF later

b. Anastomosis between pulmonary & bronchial veins: (systemic)

This will cause shunting of high pressure & congestion from the pulmonary veins to the bronchial veins.

This may lead to formation of BRONCHIAL VARICES which may rupture on coughing or straining causing severe hemoptysis.

c. Development of interstitial pulmonary barrier: (Ruptured RBCs passed & congested) → Chronic pulmonary congestion → to haemosiderosis & fibrosis.

This will decrease transudation of fluid from pulmonary capillaries. Wall become thick → ↓ Gas exchange

d. Thickening of the walls of pulmonary vessels: (interstitial lung fibrosis) → ↓ transudation. Hypertrophy of smooth muscle in the wall as the chronic lung congestion → secretion of growth factors from endothelium

Differentiation between Cardiac & Bronchial asthma

	CARDIAC asthma	BRONCHIAL asthma
Age	Any age	Usually young age
Past history	Cardiac symptoms	Chest symptoms
Duration	Short (acute LHF)	Long standing disease
Time of the attack	1 - 2 hours after sleep	Early in the morning
Sputum	Frothy, may be blood-tinged	Thick pellets (viscid)
Chest examination	Generalized wheezes, ↑ spasm Bilateral basal crepitations ↑ secretion	Generalized wheezes
Heart examination	Gallop & murmurs (HF)	Normal
ECG	Abnormal	Normal
DRUGS		
Adrenaline	Contraindicated	Improves the condition
Morphine P ₂₃	Improves the condition	Contraindicated
Aminophylline	Improves the condition	Improves the condition

Time

Chest

Heart

Vasoconstriction
inotropic effect

written

ACUTE PULMONARY OEDEMA

DEFINITION

- Rapid accumulation of fluid in the interstitial & intra-alveolar spaces of the lung.

ETIOLOGY

1. CARDIOGENIC PULMONARY OEDEMA:

Sudden increase in the pulmonary venous pressure: ^{Acute Congestion}

- Acute left sided heart failure (LSHF): e.g. AMI. (Most common cause)
- Acute exacerbation of chronic LSHF: e.g. MS with a ppt. factor as AF.

2. NON - CARDIOGENIC PULMONARY OEDEMA: ^{acute} (ARDS)

Sudden increase in the pulmonary capillary permeability:

- **SEPTICEMIA**, Pneumonia (severe), Pancreatitis.
- **STROKES**, Head injuries.
- **SHOCK**.
- **TRAUMA**, Burns.
- **TERMINAL** Liver & renal failure.
- **INHALATION** of gases (e.g. chlorine), Aspiration of gastric contents.
- **DIC**. (chemical Pneumonitis) (absent Cough Reflex)

3. OTHER CAUSES:

- Sudden expansion of a collapsed lung, e.g. rapid aspiration of massive pleural effusion.
- Severe hypoalbuminemia. (malabsorption, nephrotic ...)

CLINICAL PICTURE

1. Manifestations of the cause: e.g.

- o Typical chest pain in AMI eg chest pain (Cardiogenic pulmonary oedema).
- o Neurological manifestations in STROKE (Non - Cardiogenic pulmonary oedema).
eg hemiplegia

NB stroke: Acute Brain injury due to Vessel disease $\leftarrow$ thrombus
 embolus
 Hge

NB In any tissue 4I $\left\{ \begin{array}{l} \text{inj} \\ \text{infection} \\ \text{inflam} \end{array} \right. \Rightarrow \text{Cytokines} \Rightarrow \text{inflammatory mediators} \Rightarrow \uparrow \text{Cap. permeability}$
 + TNF- α

either by ① Haematogenously delivered to lung: eg. septicemia, pancreatitis, trauma, burns, stroke, head injury, DIC
 "tissue and endothelial inj."

or ② Locally produced in the lung in local lung lesions
 e.g. pneumonitis, inhalation of gases, aspiration of gastric content

NB in shock: $\uparrow$ Capillary permeability due to severe hypoxia
 in Liver & Renal failure: $\uparrow$ Cap. permeability due to severe toxemia
 "toxic Capillaritis"

also in Liver $\xrightarrow{\text{NO}}$ $\xrightarrow{\text{Adenosine}}$ VD
 $\star$ hypop albuminemia $\rightarrow$ $\downarrow$ Oncotic Pr.

NB Sudden expansion of a collapsed lung $\rightarrow$ $\uparrow$ -ve P_i of lung in alveoli
 $\rightarrow$ Suck BL from dilated capillaries [The Only Unilateral]

Acute Pulm. Edema

I Etiology

① Cardiogenic

- AMI (Acute LSHF)
- AF on top of MI

② Non Cardiogenic

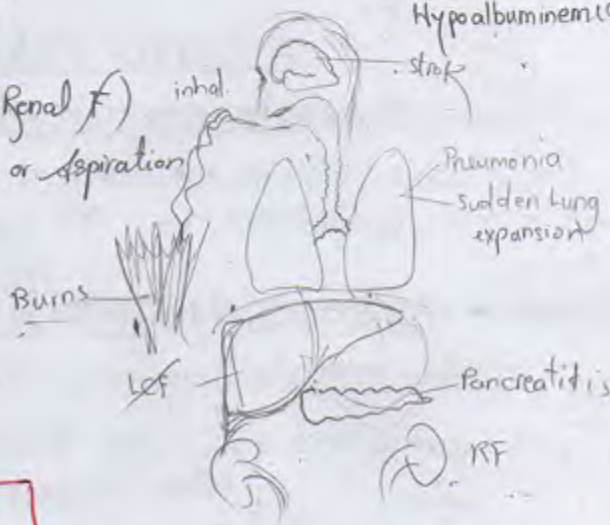
More Common: ARDS

due to Sudden $\uparrow$ in pulm. cap. permeability

- ① Sepsis, pneumonia, Pancreatitis
- ② Stroke
- ③ Shock
- ④ Trauma, Burns
- ⑤ Terminal (Liver, Renal F)
- ⑥ Inhalatⁿ of Gas or Aspiration
- ⑦ DIC

Less Common

- ① Sudden lung expansⁿ of collapsed lung (aspiratⁿ of pleural eff.)
- ② Severe Hypoalbuminemia



II C/P

② Manifest of Acute Pulm edema

- ① Manifest of Cause
- * AMI $\rightarrow$ typical chest pain
 - * Stroke $\rightarrow$ hemiplegia

- * Severe dyspnea at rest
- * Cyanosis Central
- * Generalized wheezes
- * Sweating (Tsymp.)
- * Cough (Frothy bluish sp.)
- * Generalized Coarse Bubbling crep.

III Investigations

① of the Cause

AMI $\rightarrow$ ECG & enzymes
Stroke $\rightarrow$ CT Brain

② for Acute Pulm edema

a-CXR $\leftarrow$ $\uparrow$ Vascular Mark.
Hazyiness
Kerley B Lines

b-ABG $\leftarrow$ $PO_2 \downarrow$
 $PCO_2 \uparrow$ (early)
 $\uparrow$ (late)

IV treatment

① of the Cause

AMI $\rightarrow$ reperfusion
Stroke $\rightarrow$ Antiplatelet, general care

② of acute Pulm edema

a Cardiogenic $\rightarrow$ M of HF

b Non Cardiogenic $\rightarrow$ MV
Corticosteroids
IV

2. Typical Manifestations of Acute Pulmonary Oedema:

- o Severe dyspnea at rest & orthopnea.
- o Sweating & marked irritability.
- o Central cyanosis. (hypoxia)
- o Cough with expectoration of excessive frothy blood-tinged sputum.
- 15 o Generalized BUBBLING CREPITATIONS. "massive intra alveolar edema"
- o Generalized wheezes.

INVESTIGATIONS

1. Investigations of the cause: e.g.

- o ECG & Cardiac enzymes for AMI (Cardiogenic pulmonary oedema).
- o Brain imaging (CT scan) for STROKE (Non - Cardiogenic pulmonary oedema).

2. CHEST X - Ray:

- o Exaggerated pulmonary vascular markings especially in upper lung zones. ?
- o Haziness of lung fields. (in massive edema)
- o Kerley B lines. "in lateral View" can be in "Posteroanterior" * the lower zone vessels compressed by the edema & congestion in upper zone: appear more exaggerated

3. ARTERIAL BLOOD GASES (ABG):

- o PO₂: Decreased.
- o PCO₂: Decreased at first, then, Increased late in severe cases.
Hypoxia → PRC → wash CO₂ → damaged lung due to hyperventilation

TREATMENT

1. Treatment of the cause: e.g.

- streptokinase o Reperfusion (revascularization) for AMI (Cardiogenic pulmonary oedema).
- o General care, Antiplatelets for STROKE (Non - Cardiogenic pulmonary oedema).
(aspirin)

2. Treatment of Cardiogenic pulmonary oedema:

- o Refer to the section of HEART FAILURE. P23 with

3. Treatment of Non - Cardiogenic pulmonary oedema:

- o Mechanical ventilation. (due to Respiratory failure)
- o IV Corticosteroids. → Anti inflammatory

↓
Never in Cardiogenic: salt and water retention
↳ PPT HF

COUGH

- In the cardiac patient, cough is usually due to:

- Pulmonary congestion. LSHF
- Pulmonary infection.
- Pulmonary infarction. *irritation of cough receptors in ^{lung parenchyma} pleura*

HEMOPTYSIS

- In the cardiac patient, hemoptysis is usually due to:

- Pulmonary congestion.
- Pulmonary infection. *→ delicate dilated capillaries*
- Pulmonary infarction. *] Cough → rupture delicate capillaries*

- Chronic* → • Rupture of bronchial varices.
V. acute → • Rupture of aortic aneurysm.

OEDEMA

- Pathogenesis of cardiac oedema: *LSHF: systemic congestion*

1. Increased capillary hydrostatic pressure:

- 90% of Pathogenesis*
- A) Increased venous pressure: due to stagnation of blood in systemic veins.
 - B) Salt & water retention due to:

- 1. Decreased GFR: due to ↓ renal blood flow, due to ↓ CO.

2. Increased Aldosterone: due to:

- ↑ renin release due to: reduced renal blood flow. *(Kidney)*
- ↑ stimulation of: volume receptors of Aldosterone.
- ↓ hepatic catabolism of: Aldosterone. (due to congestion)

3. Increased ADH: due to:

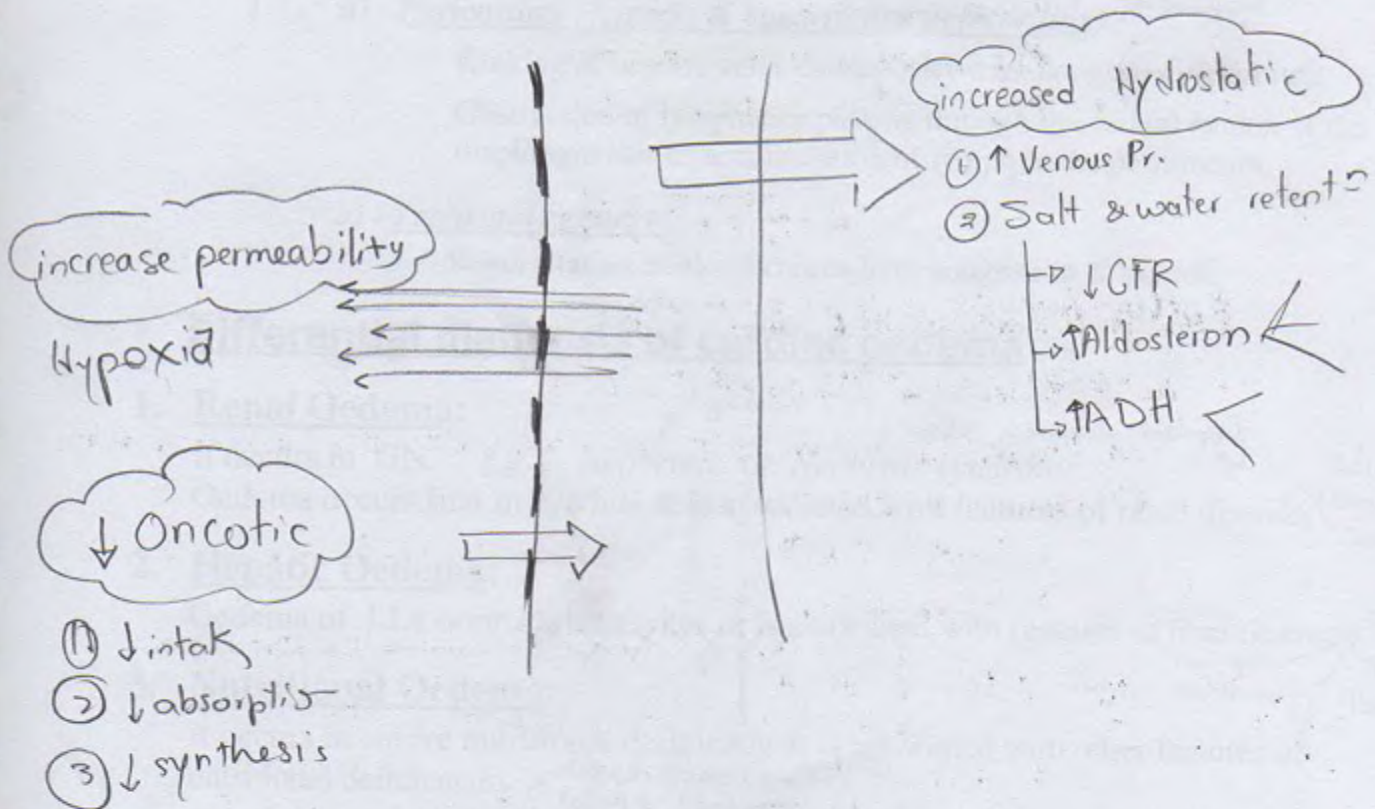
- ↑ stimulation of: volume receptors of ADH. (in atria)
- ↓ hepatic catabolism of: ADH.

2. Increased capillary permeability: due to hypoxia of the capillary walls.

↓ Albumin 3. Decreased plasma oncotic pressure due to:

- Cardiac Cachexia*
- o Decreased protein intake due to: *Congested GIT anorexia & vomiting.*
 - o Decreased protein absorption due to: *intestinal congestion.*
 - o Decreased protein formation due to: *liver congestion.*

Aortic aneurysm $\rightarrow$ Pr. ischemia on bronchial wall & become delicate
 $\rightarrow$ when it ruptures 'll rupture into the bronchus
 $\rightarrow$ bleed into bronchus
 $\rightarrow$ hemoptysis



Causes of non-pitting edema:

- 1- Myxedema (see Myxedematous hard tissue)
- 2- Lymphedema (unilateral) ~~slit~~, 3

Causes of Pitting edema: eg Nephrotic, RSLF

(NB) Causes of acute edema Nephritic, Allergic

HEMOPTYSIS

EDEMA

"Clinical description"

- Characteristics of cardiac oedema:

1. Occurs in the dependant parts of the body:

- o Ankle oedema: in ambulant patients.
- o Sacral oedema: in bed ridden patients.

* Venous stagnation:

Gravity → higher Venous Pr.

2. Bilateral:

but one side may be affected more due to:

- o Deep venous thrombosis. ^{why?} (Venous stagnation due to RSTF) ^{③ Prolonged bed Rest}
- o Postural, in patients sleeping on one side. ^{② Digitalis → ↑ coagulability}
^{④ Overdose of diuretics → dehydration → ↑ coagulability}

3. Pitting.

4. Associated with other features of cardiac disease:

example RSTF

① Neck Veins ② Congested Liver ± tender ± pulsating ± hepatogastric pulsating.

5. Oedema of lower limbs always precedes appearance of ascites:

except in two conditions in which ascites occurs first "Ascites precox":

(Pericardial tamponade)

a) Pericardial effusion & constrictive pericarditis:

← fibrous tissue

- Kinking of hepatic veins causes early liver congestion & ascites.
- Obstruction of lymphatics passing through the central tendon of the diaphragm causes accumulation of lymph in the peritoneum.

b) Tricuspid regurge:

- Regurgitation of blood causes liver congestion & ascites.

- Differential diagnosis of cardiac oedema:

1. Renal Oedema:

↑ v. gradual

→ acute v. rapidly developing

It occurs in GN, e.g. Nephrotic or Nephritic syndrome.

Oedema occurs first in eye lids & is associated with features of renal disease.

Proteinuria
haematuria
Caste

2. Hepatic Oedema:

Oedema of LLs occurs after ascites & is associated with features of liver disease:

Jaundice
gynecomastia
spider naevi
Palmer erythema
Fibrinogen
Bl. tendency

3. Nutritional Oedema:

It occurs in severe nutritional deficiency & is associated with other features of nutritional deficiencies:

Angular stomatitis (B₂)
Pellagra (B₃)
iron deficiency (nail spooning)

4. Angioneurotic Oedema:

- This **allergic** oedema occurs with constant relation to certain factors, e.g. eating certain food, or inhaling certain substances.

- **Acute onset** especially in:

Lips, Lids, Larynx, but may be generalized.

- There is usually +ve family history of:

oedema or other forms of allergy. (asthma)
other forms of allergy. (constrictive)
Leukemia

- The patient himself may have:

There is characteristic rapid response to: **anti-allergic measures.**

- Adrenaline - Antihistaminic - Corticosteroids

laryngeal edema → suffocation, shortness of breath → stridor

SYNCOPE

DEFINITION

Loss of consciousness "SUDDEN & TRANSIENT" due to acute cerebral hypoxia. There is inability to maintain postural tone & is followed by spontaneous recovery. [within 3-5 min only]

Not including
Hypoglycemic coma
traumatic coma
epileptic seizures

ETIOLOGY

1. Vasomotor syncope "Excessive Vagal Stimulation"

A) Vasovagal syncope (Neurocardiogenic syncope):

- It results from severe vagal stimulation which leads to:
Severe bradycardia, hypotension, pallor & sweating.
↳ "Exception to Mary's Law"
- It results from: Sudden severe fear, pain, trauma (e.g. to testicles or epigastrium).
- It is the most common cause of syncope & is known as simple fainting

B) Carotid sinus syndrome:

- It results from pressure on hypersensitive carotid sinus baroreceptors:
e.g. During shaving.

2. Cardiac syncope

[↓ Quantity]
(any cause of low CO) especially:

- Aortic stenosis (or any other valvular obstruction)
- Acute heart failure (e.g. AMI).
- Arrhythmias (whether tachy- or brady-arrhythmias).
- Adams-stokes attacks. [Complete Heart Block] (AVN Block)
* "attack of severe low CO"
* transient period betⁿ stoppage of 1st Purkinje focus & the other → CO restored but still low HR

3. Cerebral syncope

(reduced cerebral blood flow)
[↓ Quantity] cerebral atherosclerosis, ischemia

- Vertebrobasilar TIAs.
↳ "transient ischemic attacks"
↳ supply → RAS or Reticular formation nuclei (in Pons, medulla, midbrain)

4. Hypoxic syncope

(↓ O₂ content of the cerebral blood flow) [↓ quality]

- Fallot's tetralogy & other cyanotic diseases or severe anemia.
cyanotic spells

للتفريق بين صيغة التثنية والواحدة
 اقل من 20 months
 5 10
 5 10

(Orthostatic syncope)

- Causes include:

Defect in Sympathetic

- Autonomic neuropathy, e.g. DIABETES.
- DRUGS: sympatholytic drugs (e.g. Ganglion blockers), VD.
- Lumbar sympathectomy.

Defect in VC

- Hyponatremia. no exchange $\Rightarrow \text{Ca}^{++} \rightarrow \downarrow \downarrow \text{influx of } \text{Ca}^{++} \rightarrow \downarrow \text{sliding of actin on Myosin}$
 "Addison's disease"
 ↓
 ↓ LVF

Loss of tone

- Prolonged recumbency.
- Elderly patients. (degenerative disease)

- (Rare) syncope caused by a *variety of activities* in susceptible individuals:

- *Cough syncope* (*Tussive syncope*).
- *Micturition syncope* (*more common in old men especially at night*). BPH
- *Defecation syncope*.

- Underlying mechanism:

Straining → decreased VR → decreased CO → Syncope.

DCL = disturbed Conscious Level

All short attacks associated w/ DCL as syncope but are not due to Cerebral Hypoxia

1. **Anxiety attacks:** (panic attacks with feeling of faintness or dizziness):

- o They are not relieved by recumbency.
- o They are associated with: *anxiety, palpitation & hyperventilation.* (Hysterical)

- ## 2. Hypoglycemic attacks:

- o History: of DM with overdose of insulin or missed meal.
- o Features: of sympathetic overactivity as sweating & palpitation.

3. Epileptic attacks (Seizures): refer to Neurology.

Glucagon
Growth hormone
Catecholamine

~~Cort~~ Cortisol thyroxine

CHEST PAIN

CARDIOVASCULAR CAUSES OF CHEST PAIN:

- Myocardium:

CAD: *angina or AMI.*

- Pericardium:

Pericarditis or pericardial effusion.

- Endocardium:

Mitral valve prolapse.

- Aorta:

Dissecting aortic aneurysm.

- Pulmonary:

Acute pulmonary embolism.

- Pain of cardiac neurosis: (Psychosomatic)

- Type of patient: *neurotic patients especially young ♀.*
- Site & Reference: *Left inframamary, Localized.*
- Character & Duration: *Stitching, of variable duration.*
- Precipitating factors: *No relation to exertion.*
- Relieving factors: *Not relieved by rest.*
- Associations:

- o *Features of neurosis: especially sighing respiration.*
- o *Normal cardiac examination.*

- Huge cardiomegaly:

Rarely causes retrosternal heaviness. - *stretch of parietal pericardium*
 - *irritate mediastinal nerves*

Angina ١٥/٦/٦٠

١٠-١٢ مواد

*هستيريك
السعال الجاف*

Signs of cardiac disorders

I. GENERAL EXAMINATION

The general examination of the cardiac patient includes looking for the following signs:

1. Decubitus

- | | |
|--|---------------------------|
| 1. Orthopnea: | left-sided heart failure. |
| 2. Prayer's position (sitting up and leaning forward): | pericardial effusion. |
| 3. Squatting: | the Tetralogy of Fallot. |

2. Pallor

In cardiac disease, pallor may be due to:

1. Anemia.
2. Rheumatic activity.
3. Infective endocarditis.
4. Low cardiac output.
5. Shock.

3. Cyanosis

1. Central cyanosis, occurs in:
 - o Congenital heart disease.
 - o Hypoxic cor pulmonale.
2. Peripheral cyanosis, occurs in:
 - o Venous stagnation, e.g. *Right sided HF.*
 - o Venous obstruction.

4. Jaundice

In cardiac disease, jaundice may be due to:

1. Severe right sided heart failure.
2. Pulmonary embolism.

5. **Fever**

In cardiac disease, fever may be due to:

1. Rheumatic activity.
2. Infective endocarditis.
3. Pericarditis.
4. Myocarditis.
5. Myocardial infarction.
6. Pulmonary infarction.
7. Pulmonary infection.

6. **Clubbing of digits**

In cardiac disease, it occurs in:

1. Infective endocarditis (pale clubbing).
2. Cyanotic cardiac conditions (cyanotic clubbing).

7. **NECK VEINS**

They should be examined for:

1. Pressure: (Congested or not ??)

- It should not exceed 3 cm vertically above the level of the sternal angle.
- Normally, the neck veins are not congested.

2. Pulsations: (Pulsating or not ??)

- Normally, the neck veins are pulsating with systolic collapse.
- Normally, the pulsations are wavy & consist of:
 - a-wave: right atrial contraction.
 - x-descent: right atrial relaxation.
 - c-wave: transmitted from adjacent carotids.
upward bulge of tricuspid valve into right atrium.
 - v-wave: RA filling. (VR)
 - y-descent: RA emptying.

CLINICAL SIGNIFICANCE OF NECK VEINS

1. Normality

Flow is not compressed. A normal neck vein is easily compressible.

2. Abnormality

1. Abnormal pressure

1. Congestive heart failure

2. Pulsation

a. Right-sided HF

b. Pulmonary stenosis & aortic regurgitation

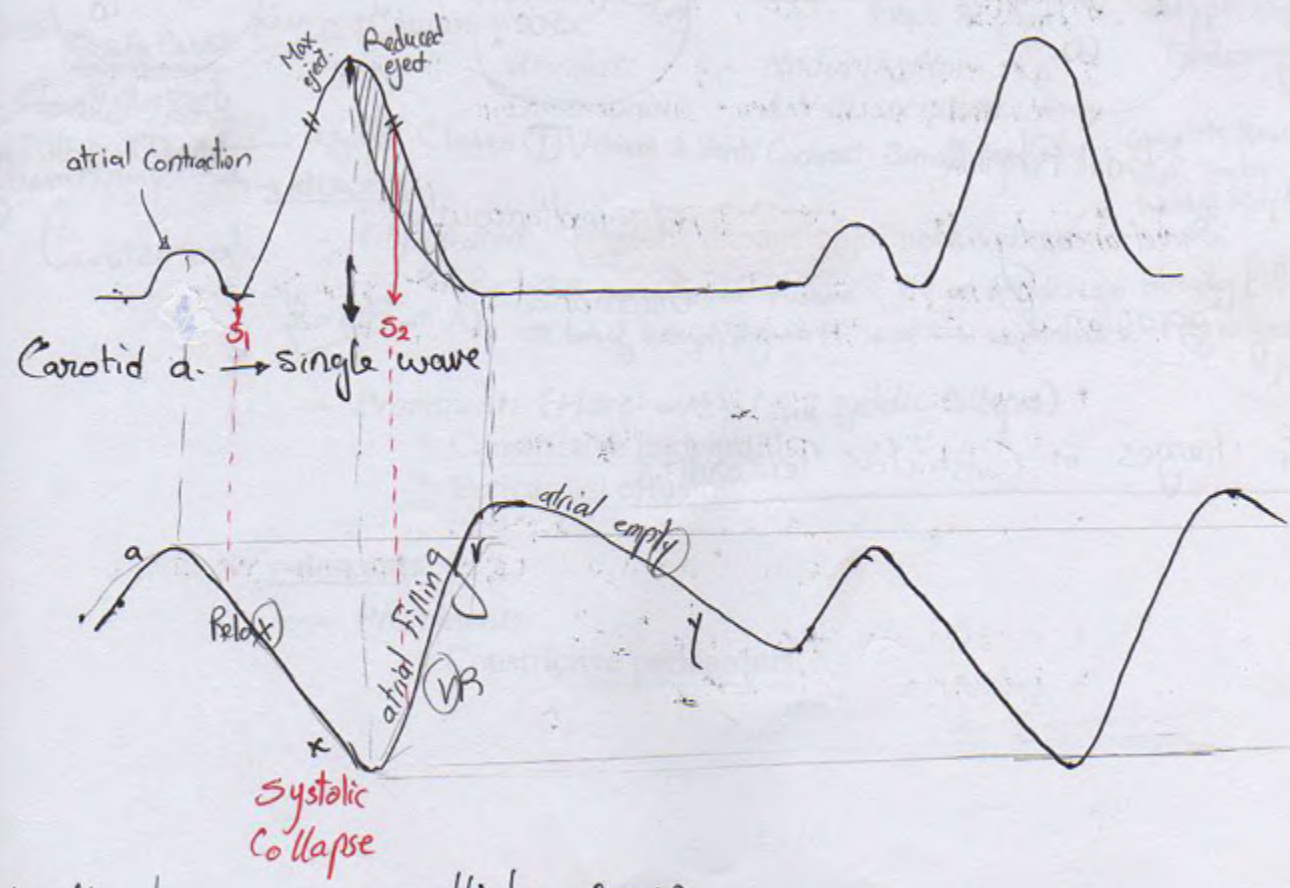
c. Hypertension

3. Non-pulsation

a. SVC obstruction, b. SVC thrombosis, c. Aortic regurgitation

4. Abnormal pulsation

Abnormal wave

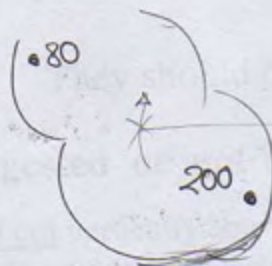


Neck Veins are → multiple waves



Nodal Rhythm

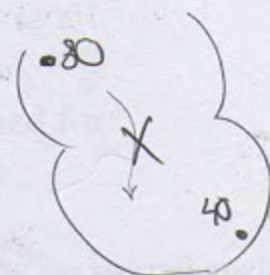
- Symultaneously
- occasional



VT

- not symultaneously
- Occasional

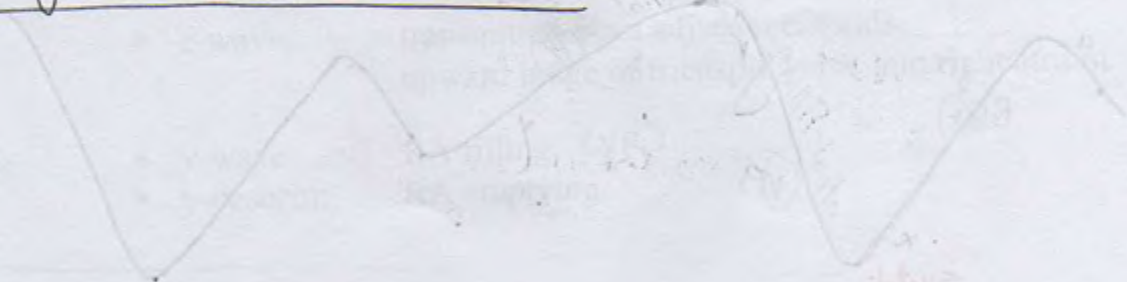
no
retrograde
Conduction



Heart Block

- not symultaneously
- occasional

Pr. changes in Constrictive Pericarditis



CLINICAL SIGNIFICANCE OF NECK VEINS:

1. Normally:

- They are non congested, & are pulsating with systolic collapse.

2. Abnormally:

1) Abnormal pressure: "Congested neck veins"

❖ Pulsating:

- Right sided HF.
- Pericardial effusion & constrictive pericarditis.
- Hypervolemia.

❖ Non-pulsating:

- SVC obstruction (SVC thrombosis or Mediastinal syndrome).

2) Abnormal Pulsations: "Abnormal waves"

❖ a- wave:

- Absent: AF. (no RA Contraction) = [absent P wave in ECG]
 - Giant: + syst. HTN → Pulmonary hypertension → PS. TS. (Strongst atrial cont.) + Hypertrophic Cardiomyopathy
 - Cannon waves:
 - Regular: Nodal rhythm.
 - Occasional: A-V dissociation.
- ↓ Filling ↓ CO ← Against Closed ⊕ atrium & Vent. Contract Simultaneously → Complete Heart Block, Vent. Tachy, Nodal Rhythm

❖ x-descent:

- Obliterated (systolic expansion) of neck veins:

- * TR. → ↑ augmented V wave (VR on Rt atrium already filled from Regurged B.L. from Rt Ventricle)
- * AF. → loss of emptying → Pr. +ve → augmented V.

— Prominent: (More -ve) (still systolic Collapse)

- * Constrictive pericarditis.
- * Pericardial effusion.

❖ y-descent:

- Prominent:
 - * Constrictive pericarditis.

8. Oedema of lower limbs

- o Right sided HF.
- o Pericardial effusion & constrictive pericarditis.

9. Abdomen

1. Hepatomegaly:

- Enlarged tender: Right sided HF & pericardial disease.
- Enlarged tender & pulsating: TR & TS.
- Enlarged tender & sharp: Cardiac cirrhosis.

2. Splenomegaly:

- Infective endocarditis (tender).
- Right sided HF & pericardial disease.

3. Ascites.

II. Cardiac Examination

A) AIM OF CARDIAC EXAMINATION:

To detect:

1. Evidence of generalized cardiac enlargement:

- Shift of the apex outwards lateral to the MCL.

2. Evidence of right ventricular enlargement:

- Apex beat: (outermost lowermost impulse)
 - Diffuse
 - Shifted outwards
 - Shows systolic retraction : accompanied by reciprocal left parasternal uplift
(**right ventricular rocking**)
- Left parasternal & epigastric pulsations.
- Dullness over the lower half of the sternum.

3. Evidence of left ventricular enlargement:

- Apex beat:
 - Localized
 - Shifted outwards & downwards
 - Shows systolic bulge: accompanied by reciprocal left parasternal retraction
(**left ventricular rocking**)
- Apex impulse:
 - Heaving (forcible , sustained): pressure overload.
 - Hyperdynamic (forcible , non-sustained): volume overload.
 - Hypodynamic (weak): myocardial disease

4. Evidence of great vessel dilatation:

- Dilated pulmonary artery:
 - Dullness & pulsations in the second left space.
- Dilated aorta:
 - Dullness & pulsations in the second right space.

5. Evidence of endocardial affection (valvular affection):

- Presence of specific murmurs & thrills.
(murmur ← على القلب)

6. Evidence of myocardial affection:

- Presence of S₃ gallop over the mitral or tricuspid areas in LV or RV affection respectively.

7. Evidence of pericardial affection:

- Refer to the chapter of " Pericardial disease "
 { Pericarditis → rub
 Effusion → dull

8. Evidence of pulmonary hypertension:

a) Precordial examination:

- Pulsations, diastolic shock & dullness in the second left space.

b) Auscultation:

4 pulm
1 (T)
palpable 2nd sound
S2
S2 is accentuated.

- Systolic ejection click. *Forcible Vent. Contraction*
- Systolic ejection murmur. *(dilated pulm. trunk)*
- Soft early diastolic murmur due to functional pulmonary regurg (Graham Steel). *(dilated pulm-Ring.)*
- S₄ over the tricuspid area. *(strong Rt atrial Contraction)*

Functional murmurs
(no organic lesions)



9. Evidence of arrhythmias:

- Change in the rate or rhythm of the heart beats.

B) TECHNIQUE OF CARDIAC EXAMINATION:

“Refer to the Clinical notes”.

THE CARDIAC CYCLE

- **First Heart Sound:**

The cardiac cycle starts by contraction of the ventricles resulting in the closure of the mitral & tricuspid valves producing the *first heart sound* (S_1).

- **Isometric Contraction Phase:**

The ventricles continue to contract while the 4 valves of the heart are closed, so the pressure inside the ventricles rises rapidly without change in the volume.

- **Ejection Phase:**

- When the pressure in the ventricles exceeds the pressure in the aorta & pulmonary artery, the aortic & pulmonary valves will open (*normally with no sound*).
- Then the blood rushes from the ventricles to the aorta and pulmonary artery, first rapidly "maximum ejection phase", then slowly "reduced ejection phase".

- **Second Heart Sound:**

After evacuation of blood, the ventricles relax, so the pressure in the aorta and pulmonary artery will exceed the pressure in the ventricles resulting in closure of aortic & pulmonary valves producing the *second heart sound* (S_2).

- **Isometric Relaxation Phase:**

The ventricles continue to relax while the 4 valves of the heart are closed, so pressure inside the ventricles falls rapidly without change in the volume.

- **Ventricular Filling Phase:**

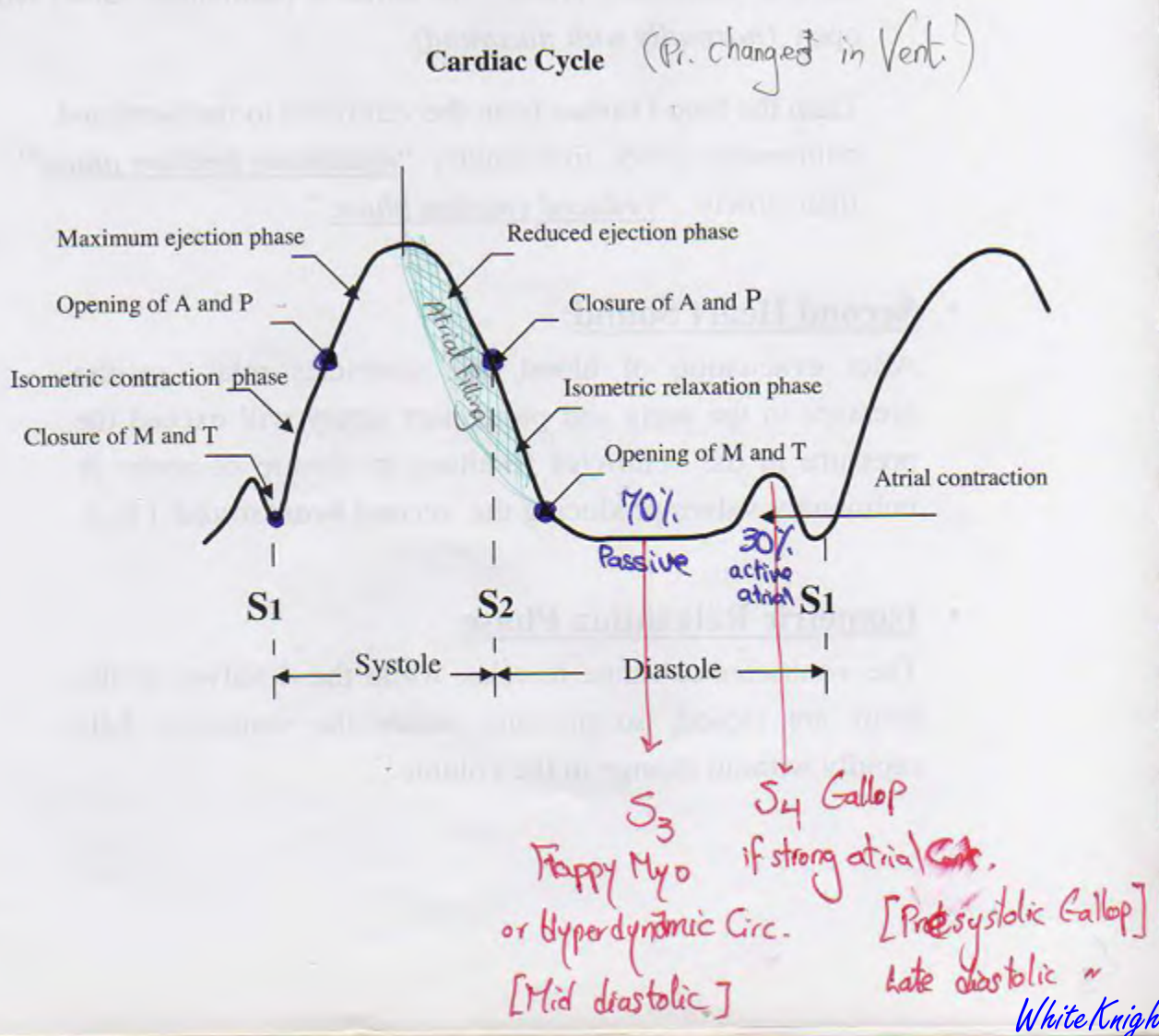
When the pressure in the ventricles becomes lower than the pressure in the atria, blood flows to the ventricles passively, at first rapidly: "maximum filling phase", then slowly: "reduced filling phase".

- **Atrial Systole:**

The last amount of blood in the atria is pushed actively by atrial contraction in the late diastole "presystole".

- **Ventricular contraction:**

Occurs again & the cycle is repeated.



* Complication of Mitral stenosis :-

* Give Reasons for .. eg accentuated S_2 in PS, —

* X-ray

* Clinical (Cardiology Cases: Valvular)

NB How to differentiate between Functional & Organic: من

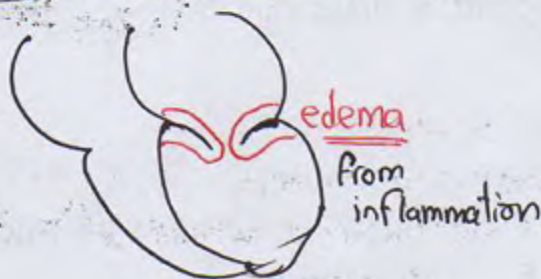
① Strength (Functional weak)

② Character (" soft)

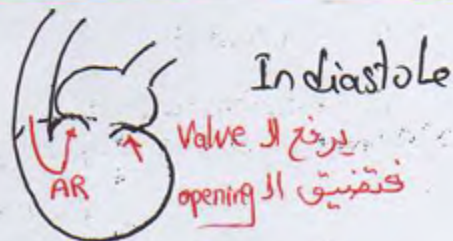
③ Thrill (" — -ve except 1 exception)

④ 1st heart sound (S_1)

Carey-Coombs in RF



Austin Flint Murmur in AR



Lt side of heart

VALVULAR HEART DISEASES

MITRAL STENOSIS

- By ECHO ↓
- Mitral valve area = $4-6 \text{ cm}^2$
 - Ant. leaflet & Posterior leaflet.

ETIOLOGY

deposition of Fibrous tissue on the ring

Organic Cause

1. Rheumatic Fever:

the most common cause.

[inflammatory] → induce fibrosis.

2. Congenital:

a rare cause.

3. Relative stenosis:

(not an organic stenosis)

المرض عكاري يعرّي

Volume overload

① Increased blood flow through the mitral valve.

② Carey-Coombs murmur in acute rheumatic valvulitis. (inflamed → edema)

③ Austin flint murmur in severe aortic regurge.

PATHOPHYSIOLOGY

- In mild cases: Less than 4 more than 2 ($2 > \text{mild} > 2$)

The blood flow through the mitral valve remains normal.

No symptoms occur.

- In severe cases: (valve area less than 2 cm^2):

The blood flow through the mitral valve is decreased. → Blood stagnant in Lt atrium → Microthrombi

Blood stagnates in pulmonary veins (pulmonary congestion).

- Later on:

Vasoconstriction of pulmonary arterioles occurs to ↓ pulmonary congestion, but this will lead to: **pulmonary hypertension.**

- Finally:

RV enlargement & then RVF will occur secondary to pulmonary HTN.

- in Pure MS: Lt. atrium is not affected (3 chambers affected)
Ventricle

Stage II

	Stage I	Stage II	Stage III	IV عاين اكتشاف
Patho-Physio- + Staging	Mild Cases $2 < \text{mild} < 4$ - Blood Flow normal - no symptoms - Fully compensated	Severe Cases $< 2 \text{ cm}^2$ - Blood Flow $\downarrow\downarrow$ - Pulm Congestion - Pulm. Venous Pr. $\uparrow\uparrow$	Later VC $\rightarrow$ $\downarrow$ pulm Congestion - Pulmonary HTN - Pulm arterial Pr $\uparrow\uparrow$	Finally RVF due to pulm HTN
c/p	asymptomatic	- pulm Congestion $\hookrightarrow$ Cough <u>dyspnea</u> hemoptesis - low CO headache, blurred vision Angina; ...	- $\downarrow\downarrow$ Pulm Congestion - 1 - $\uparrow\uparrow$ low CO symptoms	- $\uparrow\uparrow$ low CO symptoms (systemic Congestion): - confusion - Rt hypochondrial Pain - Anorexia, dyspepsia - LL edema - ascitis
Signs General	no signs	- Pulm. Congestion: - Bilateral Basal crepitations - tachypnea	① low CO: - cold pale skin - small Pulse Volume - $\downarrow\downarrow$ SBP ② Malar flush ③ Giant <u>a</u> wave	Systemic Congestion LL (edema) ascitis
Cardiac				

Signs of low CO:

- ① skin - Cold, pale cyanotic (according to severity)
- ② V/s $\leftarrow$ - Small Pulse Volume
- $\leftarrow$ - Low Systolic Blood Pressure

in Pulm HTN:

interstitial lung fibrosis $\rightarrow$ deposition of Hemosiderine

$\downarrow O_2$ $\rightarrow$ Cyanosis

$\uparrow$ Erythropoietin

redness.
Malar flush

Other Causes of Malar flush :-

- SLE
- Myxedema
- local skin diseases

Characters of Apex beat

① forcible $\rightarrow$ sustained

$\rightarrow$ non

② weak (hypodynamic)

③ Slapping (MS) (tapping) مترقش ابرو

When MS the S₁ weak:

① Double mitral $\rightarrow$ predominant regurg

② Calcified Mitral Valve

حسب اسبب و قوت $\leftarrow$ قوت بقیه بطریق (مترقش)



DM
HTN
Cancer
MS

Signs

General:

1. Stage I: no signs.
2. Stage II: signs of: pulmonary congestion. ^{Bilateral Basal Crepitations}
3. Stage III: signs of: low cardiac output, malar flush, giant a-wave. ^{tachypnea}
4. Stage IV: signs of: systemic congestion.

Cardiac:

A] Precordial examination:

Stage I & Stage II:

- Apex: normal site & slapping character. (tapping)
 - Diastolic thrill: ending in a palpable S_1 . (strong S_1)
- no Rt Vent. enlargement*
- Mid-diastolic murmur*

Stage III & IV:

- The previous findings.
- Signs of: (P_{42}) pulmonary hypertension. (stage III) ^{Pulsations}
- Signs of: right ventricular enlargement. ^{dullness}
- site of apex is shifted (stage IV) → ^{diastolic shock}
- enlarged = dilatation or Hypertrophy (compensation → stage III)

B] Auscultation:

► Stage I & Stage II

(Over the mitral area):

- MS* $\uparrow S_1$
1. Accentuated first heart sound: due to:
 - (a) Fibrosis of the mitral cusps. (Rigid Cusps)
 - (b) Forcible closure of the mitral cusps: because:
 - o They are displaced downwards due to high LA pressure. (Mechanical displacement)

NB

Diminished S_1 in mitral stenosis denotes:

- o Double mitral lesion (with predominant regurge) or
- o Calcified mitral valve. ($\downarrow$ mobility of Cusps)

2. Mitral opening snap: (بيلقح)

- It is **sharp & snapping** due to opening of the rigid cusps of mitral valve.
- It is heard in the **early diastole**:
 - o Just after S_2 (separated from it by the isometric relaxation phase).
 - o Just before the murmur of mitral stenosis.

Its presence denotes **non-calcification** of the mitral valve.

"Calcified" *مسكس الصوت لو*

3. Murmur of mitral stenosis:

- Timing:
 - Mid-diastolic presystolic with presystolic accentuation.
 - In AF, there is loss of presystolic accentuation (due to loss of atrial contraction).
- Character: rumbling.
- Site: at or slightly inside the apex.
- Propagation: not propagated.
- Position:
 - Best heard with the cone of the stethoscope.
 - In the left lateral position.

flow ↓ → ↑ turbulence

Atrial Contraction (الدوامه تزيد من)

Pr. gradient low
↓
↓ Flow
↓
no turbulence

4. Silent Mitral Stenosis (MS with no murmur) due to:

- a. High LV pressure: e.g. associated LVF.
- b. Low LA pressure: e.g.
 - Severe pulmonary hypertension.
 - RVF.
- c. In association with ASD. (Rare; in congenital cases) → ↓ LA Pr. as B. shift to RA.

► Stage III (P₄₂)

- The previous findings.
- Auscultatory findings of pulmonary hypertension:
 - a) Over the pulmonary area:
 - S₂ is accentuated.
 - Systolic ejection click.
 - Systolic ejection murmur.
 - Soft early diastolic murmur: due to functional PR. (Graham Steel murmur)
 - b) Over the tricuspid area:
 - S₄.

► Stage IV

- The previous findings.
- Auscultation over tricuspid area:
 - Pan.systolic murmur of functional tricuspid regurge.
 - RV gallop (S₃) due to RVF.

Pulm HTN

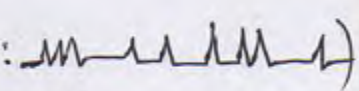
overload

hypertrophy

ischemic

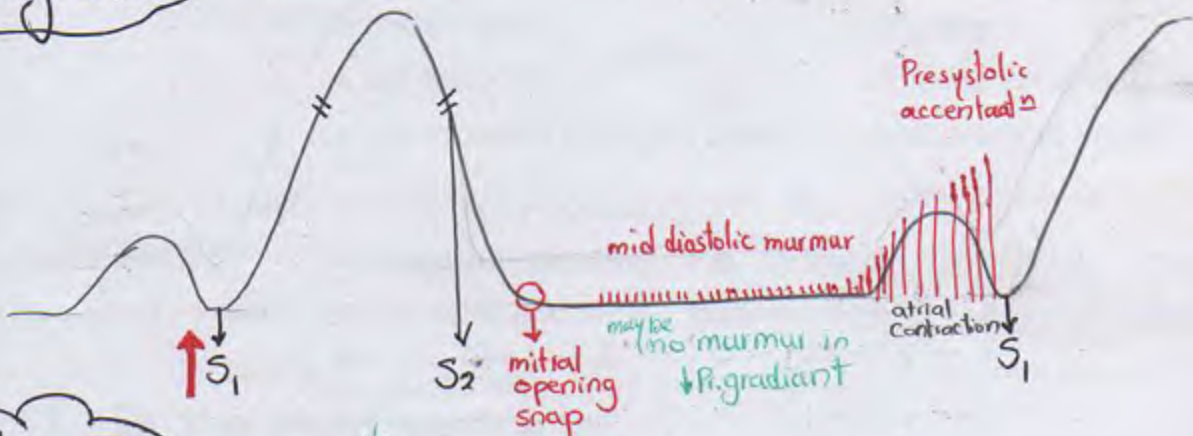
dilatation

سوال شقی: تغییرات تدریجی در مریض MS علما بعد علیہ AF
Clinical

- ① Pulse: irregular & Pulsus deficit > 10
- ② Neck Veins: absent A wave & systolic Expansion
- ③ Auscultation: loss of Pre systolic accentuation of Murmur
 - No S₄
 - S₁ Variable (due to Variable diastolic time: )
- ④ Complications: ↑ incidence of thromboembolism (30-40%)
 - acute Pulm. edema
- ⑤ ECG:
 - Absent P wave
 - irregular ECG

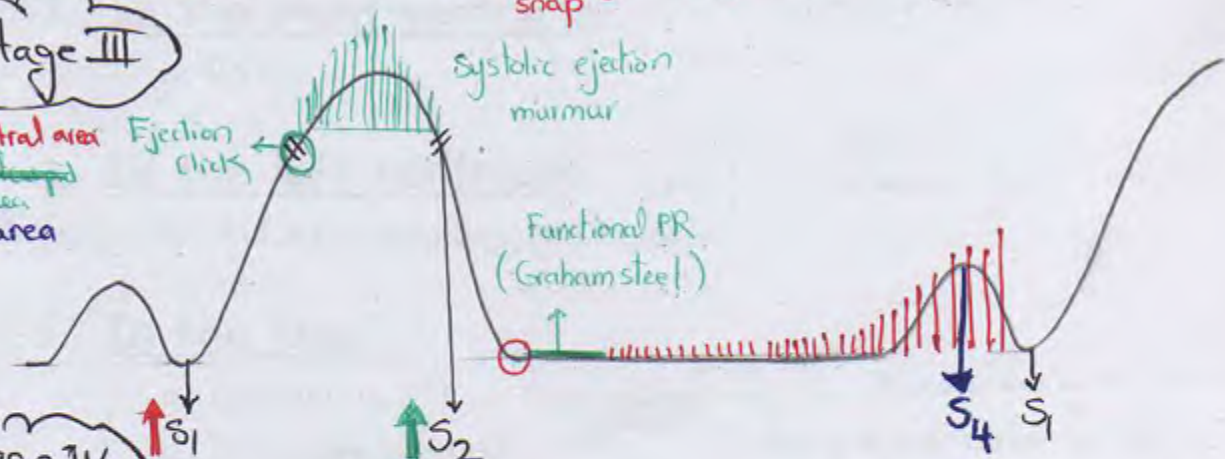
Stage I, II

→ over Mitral

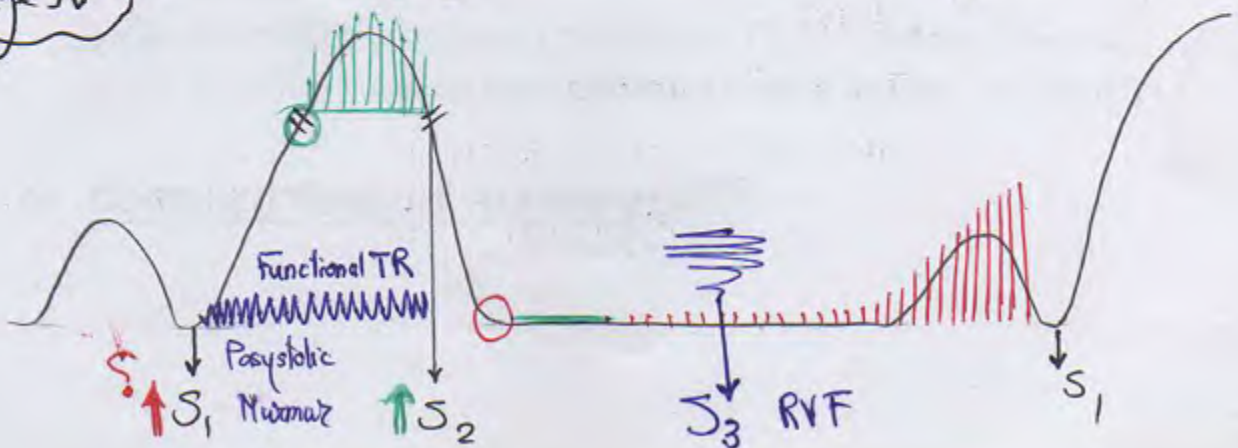


Stage III

→ Mitral area
 → Aortic area
 → T area



Stage IV



COMPLICATIONS

Written

1. In the mitral valve:

- ① o Rheumatic activity. (New attack)
- ② o Calcification. (Late) → ↓ opening snap sound
- ③ o Infective endocarditis. (Any valvular affection liable for infection → Vegetations less common than regurge)

2. In the left atrium: Stagnant bl. → stretch of fibers

- stretch → ↑ excitability
- a) Arrhythmias, especially AF. (other can happen ...)
- b) LA enlargement, causing pressure symptoms: Otner's Syndrome
- on oesophagus: dysphagia.
 - on left bronchus: dyspnea & cough.
 - on left recurrent laryngeal nerve: hoarseness of voice.

- stagnant bl. → c) Thrombo-embolic complications: in 10% of MS Patients → 30-40% if AF on top (↑ stagnation)
- RA → LV → Aorta
- Systemic embolization: e.g. cerebral, peripheral, renal.
- Ball & valve embolus: leading to syncope & sudden death.
- Mitral orifice
- Rare if the cause is Congenital ASD → PE
- من يفتح على الارجح تنظر بوجه ال

3. In the right ventricle:

- o RVF.

4. In the left ventricle:

[Only failure in DM]

- o No LVF in isolated mitral stenosis.

5. In the lung:

- o Hemoptysis. (From Pulm. Congestion) [From Bronchial Varices severe → may die]
- o Pulmonary infection. (Bl. is a good medium for infection)
- Acute Pulmonary embolism (secondary to DVT). (Prolonged Bed rest)
- Acute Pulmonary oedema is not common in mitral stenosis (see before).

6. Complications of surgery:

INVESTIGATIONS

1. CXR:

- a) Stage I: no abnormality.
- b) Stage II: (PA view & lateral view with Barium)
 - o LA enlargement. → displace esophagus & Barium / obliterated waist.
 - o Pulmonary congestion. → ↑ Bronchovascular markings
- c) Stage III & IV:
 - o Right ventricular enlargement & RA enlargement.
 - o Pulmonary hypertension. (↑ pulm. Conus)
 - o May be calcified mitral valve. حتم بيضاء المترينك حاليها

2. ECG:

- a) Stage I: No abnormality.
- b) Stage II: LA enlargement (P mitrale: broad & bifid).
 > 3 small squares
- c) Stage III & IV:
 - Right atrial enlargement (P pulmonale: tall & peaked).
 due to pulm HTN > 2.5 small squares
 - Right ventricular enlargement.

3. Echocardiography:

- The most sensitive & specific non-invasive method diagnosing MS.
- Detects the severity of stenosis by measuring:
 - o The valve area.
 - o The pressure gradient across the valve.
- Detects chamber enlargement.

4. Cardiac catheterization & angiocardiography:

- Detects the severity of stenosis by measuring:
 - o The valve area.
 - o The pressure gradient across the valve.
- Detects chamber enlargement.

Theoretical

WHEN DO WE SAY TIGHT MITRAL STENOSIS??

نظري
Theoretical

▼ Tight MS is diagnosed:

- According to the stage:

- Stage II with dyspnea more than grade II, or
- Stage III, or
- Stage IV.

يعتمد على الأعراض
والتاريخ المرضي

- According to the echocardiography:

- Valve area less than 1 cm^2 .

▼ Tight MS is an indication for SURGERY.

TREATMENT

1. Medical:

- Prophylaxis against: Rheumatic activity, Infective endocarditis (uncommon).
- Symptomatic for: Complications e.g. HF, AF, infection, embolization.

Penicillin before surgery

2. Surgical:

- Indications:

- Tight mitral stenosis (valve area is $< 1 \text{ cm}^2$).
- Marked symptoms not responding to adequate medical treatment.
- Embolization with no serious deterioration of the condition of the patient. (serious = Coma, hemiplegia → if so it is ... balloon dilatation)

- Types of operations:

- Mitral commissurotomy: closed or open.

- Valve replacement:

- By a prosthesis (tissue or synthetic).

- Indications:

- Calcification. (muffled S1)
- Associated mitral regurge. double mitral
- Recurrent stenosis after commissurotomy.

Vegetation
نمو البكتيريا على الصمام

تissue valve: Lifespan shorter,
Prosthetic valve: Lifespan longer,

smooth surface
rough surface
→ thrombi

c) Complications:

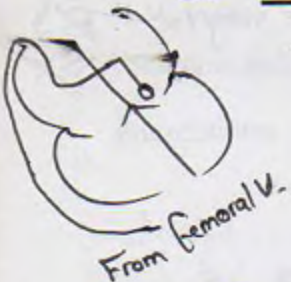
1. Embolization.
2. Arrhythmias. (Any Cardiac Manipulation)
3. Mitral incompetence. (over correction) ($\uparrow$ in closed مترشائيف)
4. Restenosis. $\rightarrow$ recurrence of fibrosis في كلا وجهي
5. Post - cardiomy syndrome: (closed)
 - Pleuro-pericarditis that may occur 10 - 15 days following the operation.
 - It is possibly an autoimmune response to damaged cardiac tissue during surgery.

6. Complications of artificial valves:

- | | | |
|---------|---|---|
| Vegit. | [| Infective endocarditis. |
| thrombi | | Thrombo-embolism. |
| Mech. | [| Mechanical dysfunction. يقفل / لا يفتح - حبيبتين لوس |
| | | Hemolytic anemia. (Mechanical destruction to RBCs : diagnosed by Blood film. $\rightarrow$ fragmented RBCs, schistocytes) |

3. Balloon dilatation:

- May be performed in some patients indicated for commissurotomy.
- e.g. $\hat{=}$ serious complications after embolization



- Important question: "Self - assessment"

"What are the changes induced by AF in a case of MS" ??

MITRAL REGURGE

ETIOLOGY

A] Organic:

1. Rheumatic fever: *the most common cause.*
2. Congenital.
3. Prolapse of the mitral valve (MVP).
4. Papillary muscle dysfunction e.g. CAD. "rupture papillary m.s."
5. Infective endocarditis. (Perforate Cusp)
6. Iatrogenic: *following mitral commissurotomy.*

B] Relative: (Functional)

- Dilatation of the mitral ring secondary to LV dilatation.

PATHOPHYSIOLOGY

1. During systole:

A part of blood regurgitates from LV to LA leading to:

- Low CO.
- LA dilation: due to increased blood volume in LA.

2. During diastole:

A large volume of blood → LV → LV enlargement which may end in LVF.

CLINICAL PICTURE

Symptoms

1. No symptoms: *in early cases.*
2. Symptoms of low cardiac output: *in late cases.*
3. Symptoms of pulmonary congestion: *in late cases.*

4. PALPITATION. Due to change of Force "Regular palpitation" "Most Common Cause" "Most Common Symptom"

Palpitation is the most common symptom

Not in AF

Signs

General:

- No signs: *in early cases.*
- Signs of low cardiac output: *in late cases.*
- Signs of pulmonary congestion: *in late cases.*

Cardiac:

A] Precordial examination:

- Signs of LV enlargement: *with hyperdynamic apex.*
- Systolic thrill: (*systolic murmur*) *over the apex.*

B] Auscultation:

1. First heart sound: **weak** (muffled) *due to failure of proper mitral closure.*
2. Third heart sound: **present** *due to excessive flow of blood from LA to LV.*
3. Murmur of mitral regurge: *even in isometric contraction phase →*
 - *Timing:* pan systolic starting with S₁.
 - *Character:* soft or harsh. (according to bl. flow & LVF)
 - *Site:* over the apex.
 - *Propagation:*
 - To the axilla. ? (in both?)
 - To the base of the heart & medially in posterior leaflet disease.
 - *Position:* heard best in the left lateral position.

• S₃ before LVF due to excessive bl. flow across valve.
 • S₃ After LVF due to excessive flow + flappy Myo.

COMPLICATIONS

Same complications of MS,

1. Infective endocarditis:
2. Left ventricular failure:

but:

is common.
occurs.

INVESTIGATIONS

1. CXR:

- No abnormality: *in early cases.*
- Enlarged LA & LV: *in late cases.*
- Pulmonary congestion: *in late cases.*
- Calcified mitral valve especially: *in late cases.*

*double mitral
stenosis
regurg*

2. ECG:

- Enlarged LA (P mitrale: broad & bifid).
- Enlarged LV.

3. Echocardiography:

- Detects the severity of mitral regurgitation.
- Detects chamber enlargement.
- Detects the cause: e.g. MVP.

4. Cardiac catheterization & angiocardiology:

- Detects the severity of mitral regurgitation.
- Detects chamber enlargement.
- Detects the cause: e.g. CAD.

TREATMENT

1. Medical:

- Same as that of mitral stenosis.

2. Surgical:

- Valve replacement.

Click murmur syndrome = Mitral Valve Prolapse 2nd Common Cause of MR
(Barlow's syndrome – Floppy mitral valve)

MCQ It is more common in young females. 5-10%.
- Mild prolapse is regarded as a normal variant. but "one of causes of chest pain"

ETIOLOGY

السبب غير معروف! Unknown, but there is familial incidence.

- Isolated abnormality, but may be associated with: Marfan's syndrome or ASD.

- 1 - MR
- 2 - AR
- 3 - dislocation lens
- 4 - ~ joint
- 5 - high arched palate
- 6 - arachnodactyly

PATHOPHYSIOLOGY

During ventricular systole, a mitral valve leaflet (commonly the posterior) prolapses into the LA. This may result in papillary muscle strain & some mitral regurge. "floppy"

Usually the case is not hemodynamically significant.

V. mild
But'll progress

CLINICAL PICTURE

Symptoms

- ASYMPTOMATIC. Most Common Presentation

"Not anginal pain" = Atypical chest pain: is the most common symptom.

- Palpitation.

Cardiac neurosis
arrhythmia
force (MR)

Strain on papillary Ms → regional ischemia
sudden traction → reflex spasm in small coronary a.
وجع القلب : Cardiac neurosis

Signs

- Mid systolic click.
- Late systolic murmur.

COMPLICATIONS

- Progressive mitral regurgitation.
- Infective endocarditis. (rough surface; deposited mucopolysaccharides)
- Arrhythmias. ① ischemia ② stretch to Ms ③ Anxiety (neurosis)
- Systemic embolization.
- Sudden death. "V. Rare" لماذا مات؟ why to die? ① Fatal arrhythmia (VF) ② Fatal embolism كل مرة على الورق

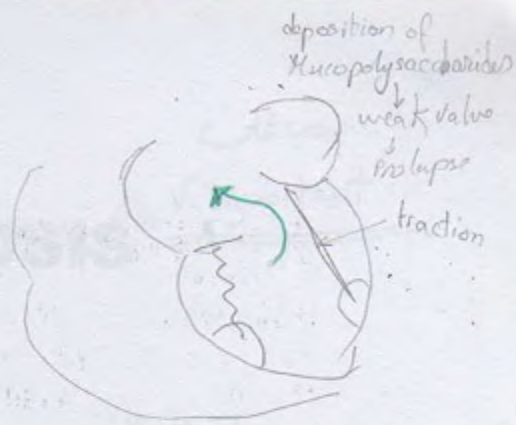
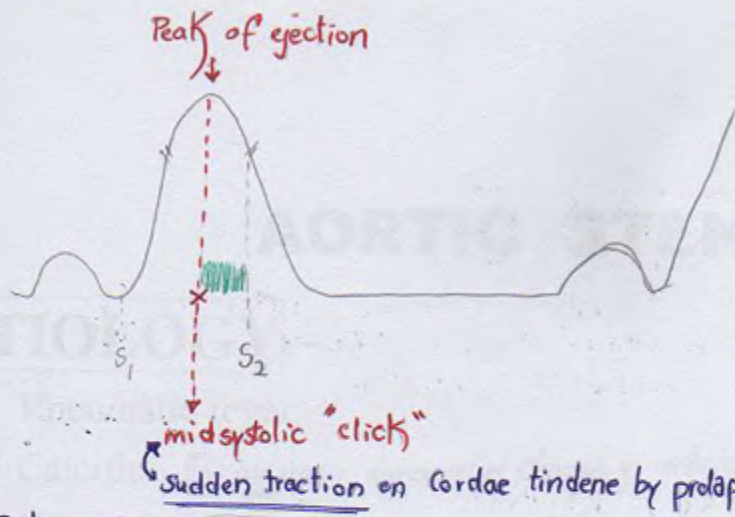
INVESTIGATIONS

Amr Allam - Echocardiography is diagnostic.

TREATMENT

- 5 • Surgery: mitral valve replacement in cases with severe MR
- ② • Prophylaxis against: infective endocarditis. (Penicillin Pre/Post operating)
- 3 • Anti - arrhythmic therapy.
- 4 • Propranolol: is effective for chest pain. Anti-ischemic (Anti-anginal)
- ① • Reassurance. (β-blocker)

مساعدة لا تجعل الوجع



Prolapse ال جمل ليا

Murmure ال رجوع يحل = late systolic

(NB) in MR = Pan systolic لانه كان مغرب من الاول

PATHOPHYSIOLOGY

CLINICAL PICTURE

Symptoms

1. No symptoms
2. Symptoms of low CO
3. Symptoms of pulmonary congestion
4. SYNCOPE

DIAGNOSIS & TREATMENT

AORTIC STENOSIS

أهم
V. Important

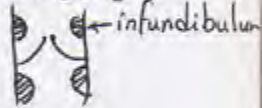
ETIOLOGY

1. Rheumatic fever.

2. Calcific. [in old age: degenerative changes in arteries eg atherosclerosis] → calcify slowly progressive

3. Congenital:

- It may be: valvular, subvalvular or supravalvular.



4. Hypertrophic cardiomyopathy (Idiopathic Hypertrophic Subaortic Stenosis; IHSS):

- It produces a subvalvular obstruction in the systole due to:

→ contraction of the hypertrophied interventricular septum. → squeeze aorta
Never digitalis → ⊕ Contraction → ⊕ Stenosis

Functional = 5. Relative stenosis

(not an organic stenosis):

a) Dilatation of the aorta:

★ Hypertension, atherosclerosis, aortic aneurysm. (eg. syphilitic)

b) Increased blood flow across the aortic valve:

Hyperdynamic circulation: e.g. AR - Anemia - Thyrotoxicosis - ...

organic → functional AS.

PATHOPHYSIOLOGY

- During systole, there is obstruction of blood flow from LV to aorta leading to:

1. Low cardiac output.

2. Pressure overload on LV leading to LVH & LVF.

- Normally, the aortic valve area is 3-4 cm². In severe AS, it is less than 0.8 cm².

CLINICAL PICTURE

Symptoms

1. No symptoms:

in mild cases.

2. Symptoms of low CO:

in severe cases.

3. Symptoms of pulmonary congestion:

due to LVF.

4. SYNCOPE:

especially exertional. (due to low CO → ↑ needs of Ms (VD) ↑ needs of Brain → syncope)

5. ANGINA:

due to:

↓ supply - Reduced coronary blood flow:

due to low CO & shortened diastole.

↑ demand - Left ventricular hypertrophy:

increases the myocardial O₂ demands.

↓ supply - Associated coronary atherosclerosis:

especially in calcific AS.

SYNCOPE & ANGINA are the most common symptoms

in Ventricular contraction against stenotic aorta

forceful sustained] Heavingness
↓ diastolic time

Signs

General:

1. Pulse:
 - Pulsus parvus et tardus (plateau pulse): *Small* *slowly* rises slowly, of small volume, returns slowly.
 - Pulsus bisferiens: *bifid pulse occurring in double aortic lesion.*
2. Systolic thrill: (Propagated from Aortic area) → over the carotid arteries.
3. Signs of low CO: in severe cases (esp. low SBP).
4. Signs of pulmonary congestion: due to LVF.

Cardiac:

A] Precordial examination:

1. Signs of LVH: with a heaving apex. *Forebode sustained* *هتفتح إيدك*
2. Systolic thrill: over second right space → apex & carotid arteries.

B] Auscultation:

- Over the aortic area:

1. Second heart sound: S_2 weak.
2. Systolic ejection click: due to opening of the rigid cusps.
3. Systolic ejection murmur: *esp. in Rheumatic Fever*
 - Midsystolic, harsh. (mid & late systolic: crescendo, decrescendo ☺)
 - Maximum over second right space → apex & carotid arteries.

- Over the pulmonary area:

- Reversed splitting of the second heart sound.

- Over the mitral area:

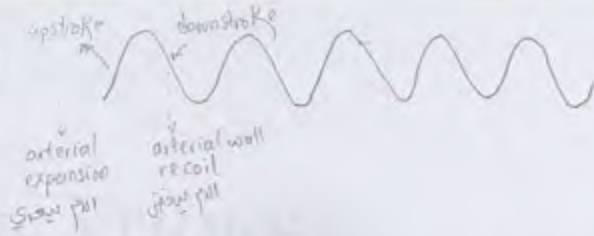
1. S_4 strong atrial contraction (elevated Pn in vent) *if tachycardia S_3 = Gallop.*
2. Propagated murmur of AS. (systolic ejection murmur)

COMPLICATIONS

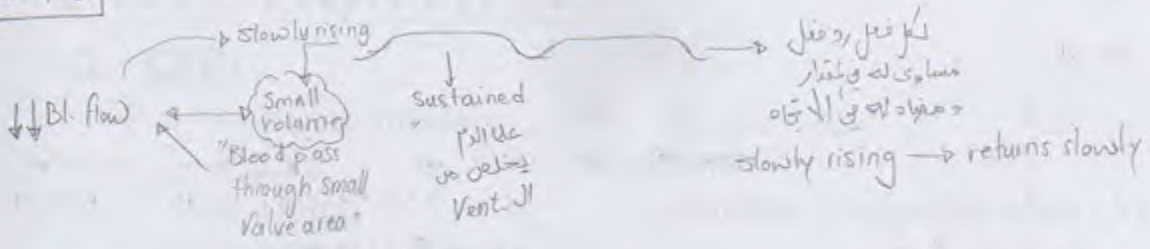
1. LVF.
2. Infective endocarditis.
3. Sudden death: usually due to VF. *arrhythmia from ischemia*
4. Heart block: in calcific AS due to extension of calcification to the AV bundle.
5. Rheumatic activity: in rheumatic AS.

القاعدة في AS هي ~ Tachycardia ~ عشان في LVF
 ماعدا في حالة حدوث AV Block ~ Bradycardia

Normal Pressure

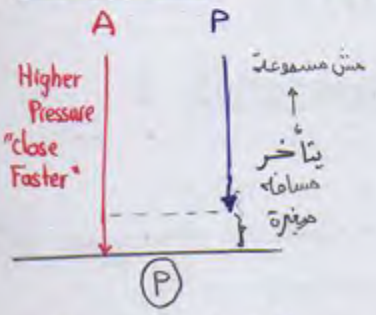


in AS

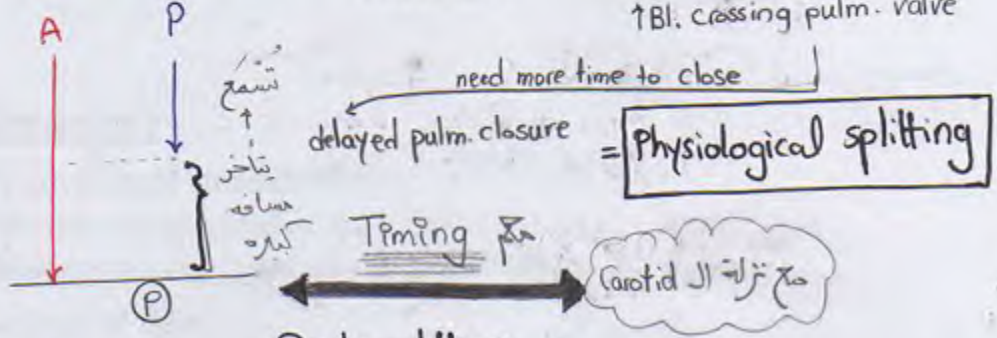


Splitting

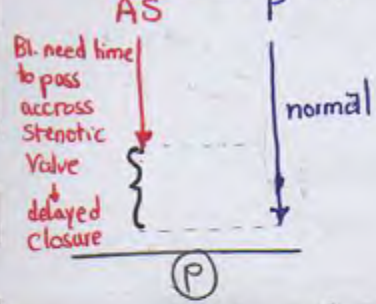
1- Normal



2- deep inspiration → ↑ Negativity intrathoracic → ↑ VR → ↑ BL. to Rt side

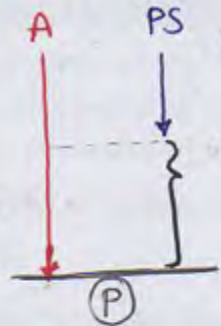


4- Paradoxical splitting = (Reversed splitting)



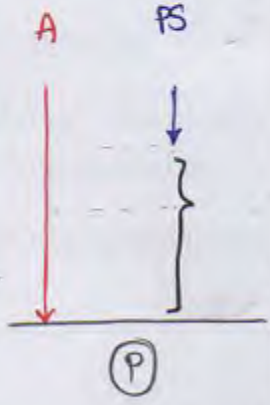
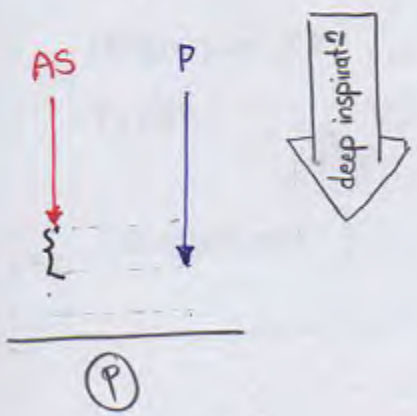
3- Pathologic = Wide splitting

مبالغة في الطبيعي

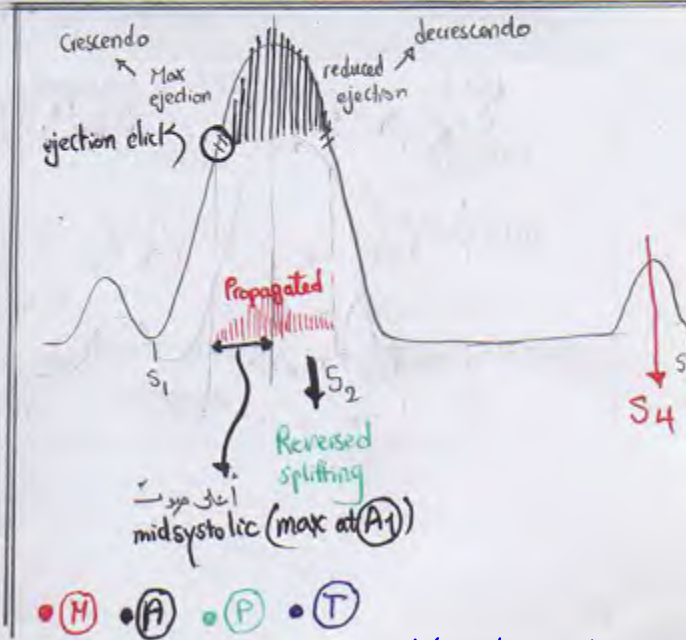


- ① RBBB: delayed impulse to Rt side → Lt Contract before Rt → Sound in Lt before Rt
- ② VSD: Bl. pass from Lt to Rt → Bl. needed to pass across aorta is less than that across pulm.
- ③ PS: Bl. need more time to pass across the stenotic valve → pulm. need more time (delayed closure)

عكس الطبيعي
(Aortic Closure delayed From pulm.)



يقال أو يكتفى
also in Angina (CAD)



INVESTIGATIONS

1. CXR:

- No abnormality: *in mild cases.*
- LV: LVH.
- Lungs: Pulmonary congestion when LVF occurs. *↑ marking*
- AORTA:
 - Small aortic knuckle or post-stenotic dilatation.
 - Aortic valve calcification may be seen. *(dense white)*

2. ECG:

- LVH.

3. Echocardiography:

- Detects the severity of stenosis by:
 - Measurement of valve area.
 - Measurement of pressure gradient across the valve.
- Detects the type of stenosis.
- Detects LVH.

MS HR : كاتشور في : *لحظة*

4. Cardiac catheterization & angiocardiology:

- Detects the severity of stenosis by:
 - Measurement of valve area.
 - Measurement of pressure gradient across the valve.
- Detects the type of stenosis.
- Detects LVH.

Clinical

	Congenital	Rheumatic	Calcific
Age	Young	Young & Middle	Old
History of RF	Absent	Present	Absent
Type	Valvular or Subvalvular or Supravalvular	Valvular	Valvular
Associations	Congenital anomalies	Other valvular lesions	Atherosclerosis

TREATMENT

1. Medical:

- Same as that of mitral stenosis.
- Anginal attacks: may be relieved by SL nitrates.

2. Surgical: (Aortic valve replacement) Indications:

1. Presence of severe symptoms. (failure of Medical)
2. Pressure gradient more than 50 mmHg. → Lt Ventricle
3. Valve area less than 0.8 cm^2 .

3. Balloon dilatation: Indications:

1. Children: with congenital AS as an alternative to surgery.
2. Elderly: with severe calcific AS who are too ill to undergo surgery.

Age	History of MI	Type	Associations
Young	Atherosclerosis	Aortic stenosis	Coronary artery disease
Young & Middle	Atherosclerosis	Aortic stenosis	Coronary artery disease
Old	Atherosclerosis	Aortic stenosis	Coronary artery disease

{NB} The Commonest Valvular diseases Causing Angina $\left\{ \begin{array}{l} \text{AS} \text{ "More Common"} \\ \text{AR} \text{ "Less"} \end{array} \right.$

TREATMENT

1. Medical

1.1. Nitroglycerin

Angina pectoris is relieved by Nitroglycerin

1.2. Beta-blockers

1.3. Calcium channel blockers

1.4. Diuretics

1.5. Statins

2. Surgical

2.1. Coronary artery bypass grafting

2.2. Valve replacement

2.3. Valve repair

2.4. Aortic aneurysm repair

2.5. Aortic dissection repair

2.6. Aortic regurgitation repair

2.7. Aortic stenosis repair

2.8. Aortic valve replacement

2.9. Aortic valve repair

2.10. Aortic valve closure

2.11. Aortic valve stenosis

2.12. Aortic valve regurgitation

2.13. Aortic valve calcification

2.14. Aortic valve sclerosis

2.15. Aortic valve stenosis

2.16. Aortic valve regurgitation

2.17. Aortic valve calcification

2.18. Aortic valve sclerosis

2.19. Aortic valve stenosis

2.20. Aortic valve regurgitation

2.21. Aortic valve calcification

2.22. Aortic valve sclerosis

2.23. Aortic valve stenosis

2.24. Aortic valve regurgitation

2.25. Aortic valve calcification

2.26. Aortic valve sclerosis

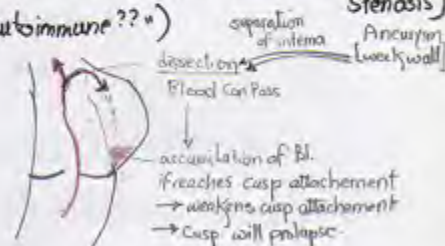
{NB} Classic Anginal pain is not nocturnal; it's exertional & not during sleep

{NB} Anginal of Lowdis; Sleep $\rightarrow$ parasympathetic pre dominance $\rightarrow$ Brady Cardia $\rightarrow$ $\uparrow$ time of each cycle $\rightarrow$ $\downarrow$ time of diastole $\rightarrow$ $\uparrow$ time of diastole $\rightarrow$ $\uparrow$ Bl. regurge $\leftarrow$ $\downarrow$ DBP $\leftarrow$ $\downarrow$ Coronary filling $\rightarrow$ Sudden wake up $\rightarrow$ $\uparrow$ sympathetic tone $\rightarrow$ tachycardia & sweating

AORTIC REGURGE

ETIOLOGY

1. Rheumatic fever: *the most common cause.*
2. Infective endocarditis. (*much more common as etiology than being a complication*) → perforate cusps
3. Congenital.
4. Dilatation of the ascending aorta as in:
 - a) Syphilitic aortitis: *syphilis produces dilatation of the aortic ring.*
 - b) Severe hypertension. → dilatation of ring. (NB: AS murmur in HTN is just turbulence not true stenosis)
 - c) Ankylosing spondylitis. (*Aortitis but due to antibodies "autoimmune"??*)
 - d) Aortic aneurysm: *with dissection.*
 - e) Marfan syndrome. (P55)



PATHOPHYSIOLOGY

- Regurgitation of blood from the aorta to the LV in diastole leads to:
 - 1) Volume overload on the LV leading to LV dilatation & later on LVF.

- 2) ↑ SBP: due to ↑ LV stroke volume. (*due to super added regurgitated blood*)
- 3) ↓ DBP: (*Rapid escape of Bl. from aorta due to regurge in diastole*) → [down to approach pressure inside Ventricle]

- ↑ SBP & ↓ DBP → wide pulse pressure → peripheral signs of AR.

(NB) Normal pulse pressure = 30-60 mmHg >60 mmHg

CLINICAL PICTURE

Symptoms

1. Generalized BODY THROBBING: due to increased arterial pulsations.
2. PALPITATION: due to forcible LV contraction.
3. Symptoms of pulmonary congestion: when LVF occurs.

4. Angina pectoris: "Two types of angina occur in aortic regurge"

- Classic angina of effort: ↓ Filling Pressure
Decreased DBP: reduces coronary filling. (*occure during diastole*)
- Angina of Lewis:
Nocturnal & associated with autonomic disturbances e.g. sweating & tachycardia.

Generalized BODY THROBBING & PALPITATION are the most Common symptoms

Signs "in any Conditions of Hyperdynamic Circulation"

المرة الوحيدة التي
G/E ال
Cardio exam.

General: one or more of "Peripheral signs of aortic regurge"

مبدأ

1. De-Musset sign: nodding of the head. دماغه يتلرز
2. Corrigan's sign: prominent carotid pulsations. رقبته تتلرز
3. Systolic thrill: thrill transmitted from Functional AS over the carotid arteries. ↑SV passing functional Carotid a. stenosis.
4. Water hammer pulse: rises rapidly, of big volume, collapses rapidly (Collapsing pulse)
5. Capillary pulsations: detected in nail bed, lips or ear lobule. [red → white → red → white] (هذا هو النبض)
6. Pistol shots: loud booming sounds heard with each pulse beat over the arteries (especially femoral) due to sudden distension of collapsed arteries.
7. Duroziez's sign:

- ♣ It consists of systolic & diastolic murmurs over the femoral artery if it is slightly compressed with the stethoscope.
- ♣ The systolic murmur is due to the rapid flow of blood to the periphery, while the diastolic murmur is due to rapid regurge of blood to the heart.

8. Blood Pressure:

a) Wide pulse pressure:

- Exaggerated difference between systolic & diastolic blood pressure.

b) Hill's sign:

- Exaggerated difference between SBP in LLs & ULs: more than 50 mmHg. [Correlation of aorta عكس]
- Normally, SBP in LLs is higher than in ULs: by about 10 - 20 mmHg.

9. Muller's sign: Cap. pulsating uvula

10. Becker's sign: in retinal Bl. vessels? (Cap. pulsations)

Cardiac:

A) Precordial examination:

- Signs of LV enlargement: with hyperdynamic apex. = forcible non sustained.
- No thrill over the aortic area: in isolated AR.

B) Auscultation:

- Over the aortic area:

1. Normal second heart sound.

Why normal S2 while supposed to be muffled?
① M. Bl. flow through valve (تكون الصوت)
② 3 cups, if 1 diseased → the other 2 are functioning

2. MURMUR of AR:

- Timing: early diastolic, decrescendo. = decreasing intensity
- Character: soft blowing. نفخة
- Site: maximum over the third space.
- Propagation: to the apex.
- Position: best heard with the diaphragm of the stethoscope, the patient is:
 - Sitting up.
 - Leaning forward.
 - Holding his breath in forced expiration.

3. Soft ejection systolic murmur:

- Due to ↑ blood flow across the aortic valve (relative AS).

NB indications of Fundus examination in Cardiology:

- ① HTN
- ② Roth spots in infective endocarditis
- ③ Becker's sign. = in retinal Bl. vessels.

NB why LL SBP > UL SBP by 10-20mmHg Normally?

- ① Bl. flow to lower limb is directly connected to the Aorta
in UL there's change in velocity during turning upwards through the aortic arch.
- ② to occlude arteries of LL during measuring BP → cuff against Bigger Mc Bulb.
(i.e. if SBP is measured by Central arterial manometer → الفرق هينقل)

Oral The only organic Murmur without thrill = AR
The only functional " with thrill = TR

Oral why AR is Best heard in leaning forward & Holding breath in forced exp.?

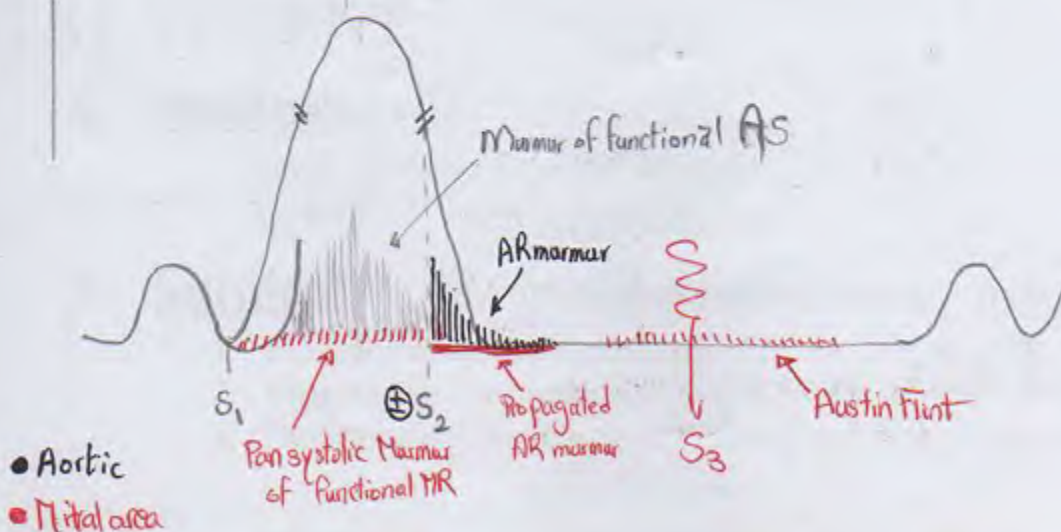
① - Leaning Forward: to Make the base nearer to the stethoscope

② Holding Breath during Expiration:

as during inspiration: ↑ negative Pressure → ↑ VR to Rt side of heart
Lung expansion → Vessels dilated
↳ pooling of blood in lung
↳ ↓ VR to Lt heart (Pulm. Vess.)

during Expiration: ↓ -ve Pr. → ↓ VR to Rt heart
squeeze pulm. bl. vessels → ↑ VR to Lt heart
↑ flow through aorta.
So murmur better heard

	Rt side	Lt side
insp.	↑ VR (↑ -ve Pr.)	distended lung vessels → ↓ VR
exp.	↓ VR (↓ -ve Pr.)	squeeze lung vessels → ↑ VR



indications of further examination in pathology

HTN

Signs of left ventricular failure in dogs and cats

General signs of congestive heart failure

1. Tachypnea & dyspnea

2. Coughing, especially at night

3. Increased jugular venous pressure

4. Pulmonary rales

5. Peripheral edema

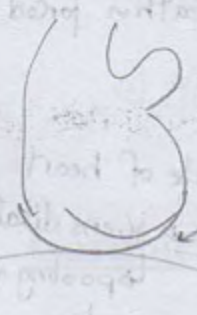
F4

RT = right thorax, RL = right leg

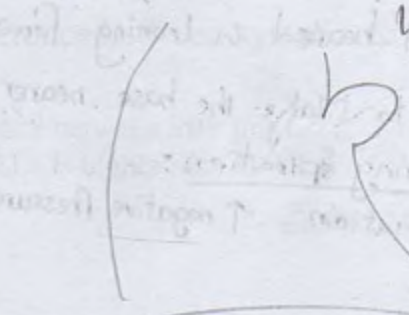
Cœur en Sabot

DR

boot shaped

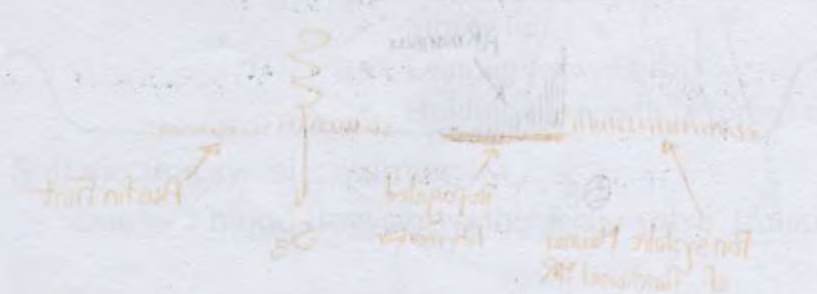


acute



During expiration, the heart is pushed up and to the right, causing the boot shape.

Normal heart shape is more rounded.



- Over the Mitral area:

1. S₃. (Volume overload + LVF)
2. Propagated murmur: of AR.
3. Pan systolic murmur: of functional MR. = Lt Vent. dilatation
4. Austin Flint murmur: (mid-diastolic) due to:

♦ Elevation of anterior leaflet of the mitral valve by the regurgitant blood from the aorta causes relative MS.

COMPLICATIONS

1. Rheumatic activity.
2. Infective endocarditis. (occurs in AR & AS)
3. LVF.

INVESTIGATIONS

1. CXR:

- LV enlargement & dilated aorta "Aortic configuration (Boot-shaped heart)".
- Lungs: Pulmonary congestion when LVF occurs.

2. ECG:

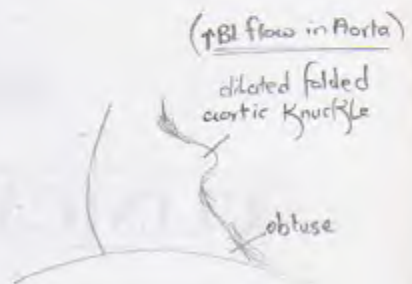
- LV enlargement.

3. Echocardiography:

- Detects the severity of the lesion.
- Detects LV enlargement.

4. Cardiac catheterization & angiocardiology:

- Detects the severity of the lesion.
- Detects LV enlargement.



TREATMENT

1. Medical:

- Same as that of mitral stenosis.
- Syphilis: anti-syphilitic ttt.

2. Surgical: (Aortic valve replacement) Indications:

1. Presence of severe symptoms. (inspite of Medical ttt)
2. Progressive cardiomegaly. (don't wait for irreversible dilatation detected by echo)
3. Declining LV functions. (↓ SF, ↓ EF detected by echo)

TRICUSPID STENOSIS

ETIOLOGY

- Rheumatic fever: the most common cause, & is almost always associated with MS.
- Carcinoid syndrome: a rare cause.

PATHOPHYSIOLOGY

- During diastole, there is obstruction of blood flow from RA to RV leading to:
 - Systemic venous congestion.
 - Low CO.

CLINICAL PICTURE

Symptoms

1. Systemic venous congestion.
2. Low CO.

Signs

General:

1. Systemic congestion including:
 - Congested pulsating neck veins with giant A wave.
 - Enlarged tender pulsating liver.
 - Ascites *before* oedema of lower limbs (ascites precox).
2. Low CO.

Cardiac:

A) Precordial examination:

- RA enlargement (dullness to the right border of the sternum).
- RV enlargement (due to frequently associated MS).

B) Auscultation:

Over tricuspid area:

- Accentuated first heart sound.
- Opening snap.
- Mid-diastolic murmur that increases by inspiration.

- oral BP measuring in IHD:-
- ① Risk factor for atherosclerosis (high Risk)
 - ② BP changes in AHTs - initial ↑ sympathetic then ↓ LF, shock, vagal Tono. Depleted BP
 - ③ ↓ BP in shock: Cardio or neuro (Difficult clinically)
 - ④ TTT Angina includes Control HTN
 - ⑤ HTN is a contraindication to Treadmill.
 - ⑥ severe HTN is C.I to streptokinase.
 - ⑦ ↓ BP is a side effect of B Blocker CCB.
 - ⑧ HTN patient w/ CAD developed AF he should be anticoagulated.

* K changes in Cardiac Disease:

- ① Drug induced hyperkalemia
- ② Drug induced hypokalemia
- ③ hypokalemia → Digitalis Toxicity
→ Torsades de pointes
- ④ hyperkalemia > 5.5 mEq/L. C.I of ACEI
- ⑤ hyperkalemia → HB
→ wide QRS
→ hyperacute T on ECG
- ⑥ patient with arrhythmias should be investigated for K changes.

INVESTIGATIONS

1. CXR:

- RA & RV enlargement.

2. ECG:

- RA & RV enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardigraphy:

- Diagnostic.

TREATMENT

1. Medical:

- TTT of right sided - HF.

2. Surgical:

- Valve replacement.

importance of corticosteroids in

① Harmful effects

- Precipitate H F
- Causes Lry HTN
- Psychosis

② Beneficial:

- in RF TTT of Rheumatic Carditis
- in pericardial effusion: ↓ inflammation & prevent CP
- AHI: Dressler's syndrome & frozen shoulder

Post Cardiotomy syndrome

- in angioneurotic oedema: anti-allergic
- in non cardiogenic acute pulmonary edema - anti-inflammatory

Painful complication of AHI

- Neurogenic shock.
- Cardiac tamponade.
- Pericarditis.

[Early] Mostly
Painless.

- Post interaction angina & recurrent infarction
- Dressler's Syndrome.
- Frozen shoulder syndrome.
- Thromboembolic complication may be painful.

"Peripheral & pulmonary

[Late] Mostly
Painful.

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TRICUSPID REGURGE

ETIOLOGY

1. Functional: the most common cause due to dilatation of the tricuspid ring secondary to RV dilatation.
2. Organic: rare:
 - Rheumatic fever.
 - Infective endocarditis.
 - Congenital.
 - Carcinoid syndrome.

PATHOPHYSIOLOGY

- During systole, blood regurgitates from RV to RA causing:
 - Low CO.
 - RA enlargement.
 - RV enlargement.
 - RVF (systemic congestion).

CLINICAL PICTURE

Symptoms

1. Systemic congestion.
2. Low CO.

Signs

General:

1. Systemic congestion including:
 - o Congested pulsating neck veins: with systolic expansion.
 - o Enlarged tender pulsating liver.
 - o Ascites before oedema of LLs (ascites precox).
 - o Mild jaundice & peripheral cyanosis (cyano-icteric face).
2. Low CO.

Cardiac:

A] Precordial examination:

- o RA & RV enlargement.
- o Rarely: systolic thrill over tricuspid area.

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- how would you treat hemodynamically stable Patient with AF & HF Dating since 2 year? → C.I for restoring Rhythm

a) in AF → slow the v. Rate. ~~X~~ ~~X~~ Digital.

8 → anticoagulation for life (2-3 INR)
→ future attack ----- unusual.
Try ablation

b) for HF TTT of HF.

- what is clinical uses of Diuretics in Cardiac Disease.

① HF → Chronic
→ acute.
→ Refractory

② HTN → Benign.
→ Urgency
→ emergency.

when are females at higher Risk of IHD

HTN
DM.
obesity
Postmenopausal.
CCBs.
hypothyroidism "Myxedematous"
Smoking

B) Auscultation: "Over tricuspid area"

1. Weak muffled S₁

2. S₃

3. Pan systolic murmur:

- Timing: pan systolic.
- Character: soft or harsh.
- Site: tricuspid area.
- Propagation: to the apex, but not to the axilla.
- Caravall's sign: murmur increases with inspiration being of right sided origin.

INVESTIGATIONS

1. CXR:

- RA & RV enlargement.

2. ECG:

- RA & RV enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardiology:

- Diagnostic.

TREATMENT

1. Medical:

- TTT of right sided - HF.

2. Surgical:

- Valve replacement.

- Before giving ACEI
 - ① Determine BP.
 - ② Chest condition
 - ③ Baseline K.
 - ④ Determine the baseline.
 - ⑤ Don't Give To pregnant female.

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PULMONARY STENOSIS

A) Organic:

- Congenital: the most common cause.
- Carcinoid syndrome: rare.

B) Functional:

- Pulmonary hypertension.

PULMONARY REGURGE

A) Functional:

- Mostly due to **PH** which causes dilatation of the pulmonary ring.

B) Organic:

- Congenital.
- Carcinoid syndrome.

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منظور من این جدول

Valve Lesion	Most important cause	Most important symptom	Most important sign	CXR	ECG
MS	Rheumatic	Pulm. Congestion esp. Dyspnea	- Accentuated S ₁ - Mid-diastolic rumbling M over apex - Pulmonary hypertension	LAE Pulm. congestion	P-mitrale P-pulmonale AF
MR	Rheumatic	Palpitation	- Pan systolic M & thrill over apex propagated to axilla	LAE LVE	LAE LVE
AS	Rheumatic Calcific Congenital	Low CO esp. Angina & Syncope	- Heaving apex - Harsh ejection systolic M & thrill " over second right space → carotid "	LVE Small aortic knuckle or Post-stenotic dilatation	LVE
AR	Rheumatic Syphilitic	Throbbing Palpitation	- Peripheral signs - Hyperdynamic apex - Early diastolic soft blowing M " over the third left space "	Boot-shaped heart	LVE
TS	Rheumatic	Systemic congestion	- Giant a-wave - Mid-diastolic rumbling M over tricuspid " ↑ with inspiration "	RAE	RAE
TR	Functional	Systemic congestion	- Systolic expansion in neck veins - Pansystolic M over tricuspid " ↑ with inspiration "	RAE RVE	RAE RVE

CONGENITAL HEART DISEASES

CRITERIA TO SUSPECT CONGENITAL HEART DISEASES:

1. **A**bsence of history of rheumatic fever.
2. **B**asal or parasternal thrills or murmurs.
3. **C**ardiac manifestations before the age of 5 years.
4. **C**entral cyanosis or systemic hypertension in infancy or early childhood.
5. **D**extrocardia without mediastinal shift.

THESE DISEASES MAY BE CLASSIFIED ACCORDING TO:

1. The presence or absence of cyanosis.
2. The hypertrophied (overloaded) ventricle.

CLASSIFICATION

The hypertrophied ventricle	Non-cyanotic	Cyanotic
1. Right ventricular hypertrophy	a) Pulmonary stenosis b) Atrial septal defect	a) Triology of Fallot b) Eisenmenger's syndrome
2. Left ventricular hypertrophy	a) Aortic stenosis b) Coarctation of the aorta c) Patent ductus arteriosus	a) Tricuspid atresia
3. Biventricular hypertrophy	a) Ventricular septal defect	a) Transposition of the great arteries b) Truncus arteriosus
4. Absence of ventricular hypertrophy	a) Any mild lesion b) Dextrocardia	a) Tetralogy of Fallot

PULMONARY STENOSIS

TYPES

1. Valvular:
 - The most common type.
 - Commonly there is post-stenotic dilatation.
2. Subvalvular: infundibular.
3. Supravalvular: the rarest.

PATHOPHYSIOLOGY

- Resistance to flow of blood from RV to pulmonary artery causes:
 - ❖ Low CO.
 - ❖ RV hypertrophy then RVF.

CLINICAL PICTURE

Symptoms

1. Low CO.
2. RVF.

Signs

General:

1. Signs of low CO.
2. Signs of low RVF.
3. Giant a-wave.

Cardiac:

A) Precordial examination:

- Signs of RV hypertrophy.
- Systolic thrill over the pulmonary area.
- Dullness without pulsations over the pulmonary area in valvular PS (due to post-stenotic dilatation).

B] Auscultation:

- Over the pulmonary area:
 - Weak S₂ with wide splitting.
 - Ejection systolic click (in valvular PS).
 - Ejection systolic murmur.
- Over the tricuspid area:
 - S₄.

COMPLICATIONS

1. Infective endocarditis.
2. RVF.
3. Pulmonary TB due to lung oligemia.

INVESTIGATIONS**1. CXR & screen:**

- RA & RV enlargement.
- Dilated non pulsating pulmonary artery (post-stenotic dilatation)
- Lung oligemia.

2. ECG:

- RA & RV enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardiology:

- Diagnostic.

TREATMENT**Medical:**

- Prophylaxis against infective endocarditis.
- Treatment of RVF.

Surgical: (In severe cases: pressure gradient more than 50 mmHg)

- Valvular stenosis: Valvotomy.
- Infundibular stenosis: Infundibular resection.

Balloon Valvotomy: may be done in: Valvular stenosis.

ATRIAL SEPTAL DEFECT

- A communication between the 2 atria.

PATHOPHYSIOLOGY

- LA pressure is higher than RA pressure, so part of blood is shunted from LA to RA which also receives blood coming from the venae cavae, leading to RA dilatation (volume overload).
- This volume overload will then pass to the RV (RV dilatation), then to the pulmonary artery (pulmonary artery dilatation), then to the lung (lung plethora).
- Later on, vasoconstrictive & obliterative changes occur in the pulmonary arterioles to decrease the lung plethora, but this will lead to pulmonary hypertension.
- Therefore RA pressure will increase till it exceeds the LA pressure causing reversal of the shunt & central cyanosis (Eisenmenger's syndrome).

CLINICAL PICTURE

Symptoms

1. Absent in small defects.
2. Dyspnea & recurrent chest infections (due to lung plethora).
3. Symptoms of RVF in late cases.
4. Symptoms of low CO in severe cases.

Signs

General:

1. Giant a-wave due to pulmonary hypertension.
2. Systemic congestion signs due to RVF.

Cardiac:

A) Precordial examination:

- Pulsations & dullness over pulmonary area.
- Signs of RV enlargement.

B) Auscultation:

- Over the pulmonary area:
 1. Accentuated S_2 (pulmonary hypertension).
 2. **Wide fixed splitting of S_2 :**
 - ❖ Wide splitting is due to increased RV blood volume which will delay the evacuation of the RV, delay closure of the pulmonary valve and delay P_2 .
 - ❖ Fixed splitting is due to the inability of inspiration to further increase the RV blood volume, because the inspiratory increase in venous return is compensated by an equivalent decrease of the left-to-right shunt, thus keeping the pulmonary blood flow constant.
 3. Systolic ejection murmur (due to increased pulmonary blood flow).
- Over the tricuspid area:
 1. S_3 (due to increased RV blood flow).
 2. Middiastolic murmur (due to increased blood flow across tricuspid).

COMPLICATIONS

1. Recurrent chest infections.
2. RVF.
3. AF.
4. Eisenmenger's syndrome : *shunt reversal*.
5. Lutembacher's syndrome : *congenital ASD & acquired MS*.

INVESTIGATIONS

1. CXR & screen:

- Dilatation of RA & RV & pulmonary artery.
- Lung plethora.
- **Hilar dance:** marked pulsations of the pulmonary artery & its branches under the screen.

2. ECG:

- RA & RV enlargement.

3. Echocardiography:

- Detects the defect.
- Detects chamber enlargement.

4. Cardiac catheterization & angiocardiology:

- Increased pressure in right side of the heart.
- Increased O_2 tension in RA than in the venae cavae.
- The catheter may pass from RA to LA through the ASD.
- Angiocardiology will show the defect.

TREATMENT

Medical:

- Prophylaxis against infective endocarditis.
- Treatment of complications.

Surgical:

- Closure of the defect.

VENTRICULAR SEPTAL DEFECT

1 SMALL VSD (ROGER'S DISEASE)

- ▼ It is a small defect in the muscular part of the interventricular septum.
- ▼ It is of no clinical significance, except that it may cause infective endocarditis.
- ▼ It is diagnosed by a loud pan systolic murmur & thrill over the left 3rd & 4th spaces.

2 BIG VSD

- ▼ This occurs in the membranous part of the interventricular septum.

PATHOPHYSIOLOGY

- LV pressure is higher than RV pressure, so part of blood is shunted from LV to RV which also receives blood coming from RA, leading to RV enlargement (volume overload).
- This volume overload will then pass to the pulmonary artery (pulmonary artery dilatation) then to the lung (lung plethora).
- Later on, vasoconstrictive & obliterative changes occur in the pulmonary arterioles to decrease the lung plethora, but this will lead to pulmonary hypertension.
- Therefore RV pressure will increase till it exceeds LV pressure causing reversal of the shunt & central cyanosis (Eisenmenger's syndrome).

CLINICAL PICTURE

Symptoms

1. Dyspnea & recurrent chest infections (due to lung plethora).
2. Biventricular failure in late cases.
3. Low CO in severe cases.
4. Palpitation.

Signs

General:

1. Giant a-wave (pulmonary hypertension).
2. Signs of biventricular failure.

Cardiac:

A) Precordial examination:

- o Biventricular enlargement with hyperdynamic apex.
- o Systolic thrill over 3rd & 4th left spaces.
- o Pulsations & dullness over the pulmonary area.

B) Auscultation:

- Over the 3rd & 4th spaces:
 - Harsh pan systolic murmur.
- Over the pulmonary area:
 - Accentuated S₂
 - Widely split S₂
 - Systolic ejection murmur.
- Over the mitral area:
 - S₃
 - Mid diastolic murmur (due to ↑ blood flow across mitral valve).

COMPLICATIONS

1. Recurrent chest infections.
2. RV & LV failure.
3. Infective endocarditis.
4. Eisenmenger's syndrome.

INVESTIGATIONS

1. CXR & screen:

- Biventricular enlargement.

2. ECG:

- Biventricular enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardiography:

- Increased pressure in RV & pulmonary artery.
- Oxygen tension is higher in RV than in RA.
- The catheter may pass through the VSD.
- Angiocardiography demonstrates the defect.

PATENT DUCTUS ARTERIOSUS

- Persistence of fetal communication between the left pulmonary artery & the aorta, just distal to the left subclavian artery.

PATHOPHYSIOLOGY

- Since the aortic pressure is higher than the pulmonary pressure in both systole & diastole, blood will be shunted from aorta to pulmonary artery in both systole & diastole, leading to:
 - Increased pulmonary flow causing pulmonary artery dilatation.
 - Increased LA flow causing LA enlargement.
 - Increased LV filling causing LV enlargement.
 - Increased LV SV causing increased systolic BP.
 - Diastolic shunt of blood from aorta to pulmonary causing decreased diastolic BP.
 - Later on, pulmonary hypertension & reversal of the shunt occur.

CLINICAL PICTURE

Symptoms

1. Dyspnea & recurrent chest infections (lung plethora).
2. Symptoms of LVF in late cases.

Signs

General:

1. Big pulse volume & water-hammer pulse.
2. Giant a-wave due to pulmonary hypertension.
3. Differential cyanosis (cyanosis only in lower limbs with shunt reversal).

Cardiac:

A) Precordial examination:

- LV enlargement with hyperdynamic apex.
- Continuous thrill over the left infraclavicular area.
- Pulsations & dullness over the pulmonary area.

B) Auscultation:

- Over the left infraclavicular area:
 - Gibson's murmur: continuous machinery murmur propagated to the pulmonary area.

- Over the pulmonary area:
 - Accentuated S_2 .
 - Reversed splitting of S_2 .
- Over the mitral area:
 - S_1 .
 - Mid diastolic murmur.

COMPLICATIONS

1. LVF.
2. Infective endocarditis .
3. Eisenmenger's syndrome.

INVESTIGATIONS

1. CXR & screen:

- LA & LV enlargement.
- Dilated pulmonary artery.
- Lung plethora.

2. ECG:

- LA & LV enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardiology:

- Diagnostic.

TREATMENT

Medical:

- Prophylaxis for infective endocarditis.
- Treatment of complications.

Surgical:

- Closure of the ductus.

COARCTATION OF THE AORTA

- Congenital narrowing in a part of the descending aorta .

CLINICAL SIGNIFICANCE

- It is an important cause of secondary hypertension.

COMPLICATIONS

1. Secondary hypertension.
2. LVF.
3. Infective endocarditis .
4. Dissection or rupture of aorta.

DIAGNOSIS

- Harsh systolic **murmur** over the left interscapular area due to passage of blood in the coarctation.
- **Rosler's sign:** (on CXR)
Notching of the lower margins of the ribs, due to their erosion by the dilated collaterals.
- **Echocardiography:** is diagnostic.

TREATMENT

Medical:

- Prophylaxis for infective endocarditis.
- Treatment of complications.

Surgical:

- Balloon dilatation.

TETRALOGY OF FALLOT

IT CONSISTS OF

1. Pulmonary stenosis (infundibular type).
2. Ventricular septal defect (big).
3. Overriding aorta.
4. Slight RV hypertrophy.

PATHOPHYSIOLOGY

Pulmonary stenosis results in:

1. Decreased pulmonary blood flow.
2. Increased RV pressure leading to:
 - a) Right to left shunt of blood across the VSD.
 - b) RV enlargement, but only of slight degree, why? Because the presence of the big VSD prevents excessive rise of right ventricular pressure (the blood in the RV has 2 ways to leave the ventricle).
3. Most of the deoxygenated blood passes from both ventricles into the overriding aorta leading to central cyanosis & only a small part passes through the PS to the lungs to get oxygenated.

CLINICAL PICTURE

Symptoms

1. **C**entral Cyanosis: Since birth.
2. **C**yanotic spells: " Syncopal attacks "

Due to spasm of the hypertrophied infundibulum forcing all the deoxygenated blood in the RV to the aorta leading to cerebral hypoxia, syncope, convulsions & more cyanosis.
3. **S**hortness of breath: exertional dyspnea.
4. **S**quatting position: improves the pulmonary blood flow & thus improves dyspnea because:

kinking the femoral arteries will increase the resistance to the blood flowing through the aorta, & thus forcing more blood to pass through the pulmonary artery to the lungs.
5. **S**tunted growth.

Signs

General:

1. Central cyanosis.
2. Clubbing of the fingers.

Cardiac:

A) Precordial examination:

- Slight RV enlargement (may be absent).

B) Auscultation:

1. Second sound:

- Single: aortic component only.
- Accentuated : due to:
 - Increased aortic blood flow.
 - Aorta is anteriorly positioned.

2. Systolic ejection murmur (maximum in 3rd left space): due to:
 - *Infundibular stenosis.*

COMPLICATIONS

1. **P**olycythemia, due to hypoxia.
2. **P**ulmonary TB due to lung oligemia.
3. **P**aradoxical embolism.

INVESTIGATIONS

1. CXR & screen:

1. Coeur en sabot:

- ♦ Dilated aorta.
- ♦ Small pulmonary artery (exaggerated waist).
- ♦ RV enlargement (uplifted apex).

2. Lung oligemia.

2. ECG:

- RV enlargement.

3. Echocardiography:

- Diagnostic.

4. Cardiac catheterization & angiocardiology:

- ☑ Increased pressure in RV & decreased in pulmonary artery.
- ☑ Decreased O₂ tension in aorta.
- ☑ The catheter may pass from the RV to the aorta.
- ☑ Angiocardiology ; will show the anomaly.

TREATMENT**Medical:** " For cyanotic spells "

- O₂ inhalation
- β -blockers or verapamil (they prevent & treat the attack by relaxing the infundibulum).

Surgical:

- Complete correction.
- Infundibular resection (Brock's operation).

TRIOLOGY OF FALLOT**IT CONSISTS OF**

1. Pulmonary stenosis (valvular type).
2. ASD.
3. Marked RV hypertrophy.

CLINICAL PICTURE

- Features of valvular pulmonary stenosis plus central cyanosis appearing 2 years after birth.

INVESTIGATIONS

1. CXR:

- As PS.

2. ECG:

- As PS.

3. Echo and Doppler:

- Diagnostic.

4. Catheterization:

- Diagnostic.

TREATMENT

Medical:

- For prophylaxis and treatment of complications.

Surgical:

- Complete surgical correction.

	Fallot's Tetralogy	Fallot's Triology
- Cyanosis	Since birth	Later on
- Squatting	Common	Absent
- Neck veins	Normal	Giant "A" wave
- Arachnodactyly & high arched palate	Absent	Common
- RV enlargement	Slight	Marked
- Pulmonary area	Resonant	Dull
- Thrill	Uncommon	Common
- Second HS	Single & accentuated	Widely split & weak
- Ejection click	Absent	Present
- Gallop	Absent	Common

DISEASES OF THE PERICARDIUM

1. Dry pericarditis. → inflam w/out effusion
2. Pericardial effusion. → effusion
3. Constrictive pericarditis. → fibrosis bet 2 layers of pericardium
4. Adhesive pericarditis. → fibrosis in pericardium adherent to its surrounding structure

DRY PERICARDITIS

ETIOLOGY

1. Idiopathic (probably viral: Coxsackie B or Echo virus): the most common cause
 2. **Infections:** Viral, Bacterial, TB.
 3. **Inflammation:** FMF. (familial mediterranean fever)
 4. Immunological: SLE, RA. inflam by Ab (auto immune)
 5. Iatrogenic: INH, Hydralazine, Procainamide. → Part of lupus like arthritis, skin rash, Pericarditis, pleuritis
 6. Irradiation. e.g for Cancer
 7. Renal failure. (Dry or effusion never constrictive)
 8. Rheumatic.
 9. Post-traumatic.
 10. Post-cardiotomy syndrome.
 11. Myocardial infarction. (early & late: Dressler's sign)
 12. Malignant infiltration. esp. leukaemia
Rare
- Case: Renal failure & auscultation of pericardial rub = pericarditis
So patient, need Dialysis

CLINICAL PICTURE

Symptoms

1. Symptoms of the CAUSE. N/S SLE renal failure
2. GENERAL symptoms: e.g.
- Fever, malaise, headache.

3. LOCAL symptom: "CHEST PAIN" → Pericardium is pain Sensitive

- It is due to inflammation of both the pericardium & the adjacent pleura.

↳ pleura & pericardium are adherent

* why radiate to shoulder?

• Due to irritation of phrenic n. which runs beside heart in middle & inferior mediastinum

• phrenic n. irritate dermatomes in shoulder

• Site & Reference:

Precordial & may radiate to the shoulders.

• Character & Duration:

Severe Stitching, Continuous.

• Increases by:

Inspiration & movements of chest wall.

• Decreases by:

Sitting up & leaning forward. (↓ friction)

• Associated symptoms:

May be pleuritic chest pain.

↳ stitching pain ↑ w/ insp & cough

Signs

1. Signs of the CAUSE.

2. GENERAL signs:

e.g.

- Fever.

3. LOCAL sign:

"PERICARDIAL RUB"

↳ rough

- It is due to friction between the 2 layers of the inflamed pericardium.

- Site: All over the heart. Best heard over the Base of the heart & the Bare area.
- Timing: All through cardiac cycle. Most obvious in systole, but may also be heard in diastole.
- Character: Scratching
- Increases by: Pressing the stethoscope against the chest wall.

Complications

all Dry
Pericarditis

→ Pericardial effusion. "accumulation of inflammatory exudate"

INVESTIGATIONS

1 ECG:

• Elevated ST segment.

• Flat or inverted T waves.

also effect not toxicity of digitalis

2 Investigations of the cause.

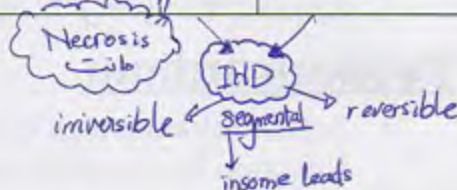
RF → urea, Creatinin (RFT)
SLE → Ab (serology)

DIFFERENTIAL DIAGNOSIS

1. DD of: the causes of dry pericarditis. *کلیتین غیر سبب ال ۱۲ سبب ال مایٹ*
2. DD of: the causes of acute chest pain. eg ...
3. DD of: the causes of elevated ST segment. *QAMI ② Variant angina ③ Late compl. of MI in Myocardial aneurysm*

DD of elevated ST segment

	Acute pericarditis	AMI	Prinzmetal angina
<i>Elevated ST</i>	Concave upwards	Convex upwards	Flat
<i>Elevated ST</i>	In all leads (<i>diffuse disease</i>)	In some leads	In some leads
<i>Pathological Q wave</i>	Absent	Present	Absent
<i>Cardiac enzymes</i>	Normal	Elevated	Normal



TREATMENT

Symptomatic:

- Analgesics & Anti-inflammatory drugs for the pain.

Specific:

- Treatment of the cause. eg RF → Dialysis, SLE → Corticosteroids

The most important points in DRY PERICARDITIS

- Viral pericarditis is the most common cause (Coxsackie B & Echo).
- One of the most important causes of acute chest pain.
- Pericardial rub.
- One of 3 important causes of elevated ST segment in ECG.

PERICARDIAL EFFUSION

ETIOLOGY

→ accumulation of fluid in pericardial space (↓ reserve volume in pericardium)
→ classification after diagnostic tapping → nature of effusion

1. Seropericardium

(ACCUMULATION OF EXUDATE)

► It occurs in all conditions that cause dry pericarditis especially: Any Cause of the 12

الانس TB:

- Malignancy.
- Viral.
- Rheumatic.

“the most common cause of pericardial effusion”.

► Hemorrhagic pericarditis is a severe form of seropericardium with excessive RBCs:

- TB. (Caseation & necrosis + erosion of bl. vessels)
- Myocardial infarction. (necrotic tissue → hemolysis of bl. RBCs)

• Renal failure. (Bleeding tendency) (toxins coat platelets)

- Malignancy. (Leukemia is the malignancy of pericardium: ① erosion B.V. ② tissue necrosis stage ③ neo-vascularization)

2. Pyopericardium

(ACCUMULATION OF PUS)

- Severe inflammation of the pericardium due to:
Infection by pyogenic organisms, e.g. Staph. & Strept.

indicate therapeutic tapping

3. Hydropericardium

(ACCUMULATION OF TRANSUDATE)

- H Heart failure. (systemic Congestion)
- H Hepatic failure. (1st Hypoproteinemia: 1st site is ascites due to associated portal HTN)
- H Hypoproteinemia, e.g. Nephrotic syndrome & liver failure: 2nd site eyelids
- H Hypothyroidism. → ↑ capillary permeability (H₂O in capillary wall) → ↑ cholesterol (crystals in pericardium → absorb H₂O)

4. Hemopericardium

(ACCUMULATION OF BLOOD)

► The pericardial fluid is frank blood:

- Rupture of the heart: trauma or infarction.
- Rupture of the coronaries: trauma during catheterization.
- Rupture of: dissecting aneurysm of the aorta.
- Hemorrhagic blood diseases. يترافق بسهولة

Emergency
Rate of accumulation is v. rapid

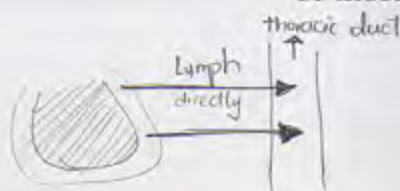
5. Chylopericardium

(ACCUMULATION OF LYMPH)

Mediastinal synd. → Obstruction:
Filaria → Rupture:

of thoracic duct. (back P.) (mediastinal syndrome)
of thoracic duct. (inj to duct & pericardium)

milk white



NB Thermost Common Cause of:

Dry pericarditis → Coxi, echo

Pericardial Effusion → TB

Pleural Effusion → TB

Peritoneal Effusion → LIVER disease (ascites)

Constrictive pericarditis → TB

CLINICAL PICTURE

Symptoms

1. Symptoms of

2. Symptoms of

3. Pericardial pain

• Onset: acute pain

• May be referred

4. Pressure symptoms

• Dyspnea

• Dysphagia

5. Symptoms of the

PATHOPHYSIOLOGY

- The manifestations depend on:

- Amount of fluid.
- Rate of accumulation.

- Cardiac tamponade (severe compression) may occur in severe cases when:

- The amount of fluid: fills the pericardial reserve volume. *→ Compression on heart → tamponade*
- The rate of accumulation: exceeds the rate of stretch of the parietal pericardium.

- The amount of fluid necessary to produce cardiac tamponade may be:

- (Rate) • As small as 200 ml when the fluid collects rapidly. (e.g. haemopericardium)
- (amount) • Over 2000 ml when it collects slowly (allowing adaptation of the pericardial sac) (e.g. TB)
→ Max. elastic limit.

- Cardiac tamponade causes ↓ cardiac relaxation & ↓ cardiac filling:

- الاعراض* { 1. Systemic congestion: due to stagnation of blood behind the heart. (Can't adapt to accept VR "Can't Relax")
2. Low CO. *impaired diastolic function (late diastole & early diastole) الاثر*
- All through the diastole*

CLINICAL PICTURE

Symptoms

1. Symptoms of: Systemic congestion. { *اضيق*
2. Symptoms of: Low CO.
3. Precordial pain:
 - Dull aching pain: due to stretch of the parietal pericardium.
 - May be referred to: the shoulder along the phrenic nerve.
4. Pressure symptoms: e.g.
 - Dyspnea: improves by sitting up & leaning forwards (prayer's position).
 - Dysphagia: due to compression of the oesophagus.
5. Symptoms of the cause: e.g. TB toxemia.
 - * night sweat, night fever
 - * loss of weight, loss of appetite

Signs

1. Signs of the CAUSE.

e.g. TB.

apical ← Fibrosis
Cavitation

2. GENERAL signs:

I. Decubitus: Prayer's position.

II. Signs of: Systemic congestion:

a) Neck veins:

- Congested pulsating neck veins.

- Kussmaul's sign: (inspiratory filling)

Due to failure of the right side of the heart to accept the increased venous return during inspiration.

- Attenuated or absent y descent. [atrial emptying due ↓ filling]

ليه تنخفض ببطء؟

b) Enlarged tender liver.

c) Ascites before oedema of lower limbs

(ascites precox)

III. Signs of: Low CO:

a) Low SBP, weak pulse, pallor. (skyn, Bl. Pr.) (⊖ filling → ↓ CO)
Pulse

b) PULSUS PARADOXUS: (↓ pulse volume during inspiration)

- Description:

- It is a misnomer as it is an exaggeration of the normal pattern & not a paradox.
- In normal subjects, during inspiration, the lungs expand & accommodate more amount of blood, thus there will be a normal ↓ SBP (less than 10 mm Hg), and a ↓ pulse volume.
- In pericardial effusion, during inspiration, there will be an exaggerated ↓ SBP (more than 10 mmHg) & an exaggerated ↓ in pulse volume... * WHY ??

- Detection:

- It can be detected by palpation of the arterial pulse, or by detecting ↓ SBP (more than 10 mmHg) during inspiration using a sphygmomanometer.

- Value:

- It is an important clue to the presence of cardiac tamponade.

بتمنى بدل ما يتفجى
المفرغ من الدم من الوريد
يحب الدم من الوريد
كان بيحط الدم

- **Explanation:** ¹

Since both ventricles (*in cardiac tamponade*) share a tight fixed incompressible covering, i.e., the pericardial sac, the inspiratory enlargement of the RV compresses and reduces LV volume; leftward bulging of the interventricular septum further reduces the LV cavity as the RV enlarges during inspiration. This will cause an exaggerated reduction in the LV stroke volume during inspiration.

- **Other causes of pulsus paradoxus include:**

- Cardio (• *Constrictive pericarditis. (tamponade)*)
- *Restrictive cardiomyopathy.*
- chest (• *COPD.*)
- *Severe bronchial asthma.*
- *Pulmonary embolism. انشغاض رئوي*

3. **CARDIAC signs:**

A] **Inspection & palpation:**

- Apical pulsations: weak or absent.
- Precordial bulge: may occur in children.

B] **Percussion:**

- Dullness to the right border of the sternum.
- Dullness outside the apex.
- Dullness on the pulmonary area (shifting dullness). ————— (resonant - قلوب)
- BARE AREA: ↑ size + stony dullness.
- **EWART'S SIGN:** dullness over the left lung base due to its compression collapse by the effusion.

C] **Auscultation:**

- Muffled heart sounds. طيفه غزل

COMPLICATIONS

1. **Cardiac tamponade:** severe compression causing ↓ BP, ↑ JVP, HF or Shock. Congested neck V.
2. **Constrictive pericarditis.** *exudate (inflammatory)* *low CO shock* *diastolic*

Beck's Triad:

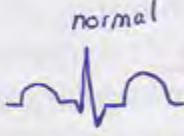
↓ BP, ↑ JVP, Muffled heart sounds.

¹ Harrison's principles of Internal Medicine "15th edition, 239 Pericardial disease"

even
in TB Corticosteroids under cover of antibiotics

INVESTIGATIONS

I. ECG:

- Low voltage.  →  Low Voltage
- Diffuse nonspecific ST-T changes (Depressed ST, inverted or flat T). 

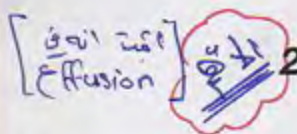
↓ motility
Barrier of fluid

II. IMAGING:

1. CXR:



- ① Symmetrical ↑ of cardiac shadow with smooth borders & loss of angulations (Flask-shaped).
 ie no pailm Conus no waist of heart
- ② Cardiophrenic angle may be obtuse (due to shadow of collapsed lung) (Rotch's sign).
 in Pericardium was Ewart's sign
- ③ Change of the shape of the heart with change of position. (shifting dullness)
- ④ Change of the size of the heart from day to day. (↑ worsening or ↓ better)
- ⑤ Double contour of the cardiac borders.
 different degree of Contrast betw cardiac tissue line & effusion line
- ⑥ Diminished cardiac pulsations under the screen. (limited movement by effusion)
- ⑦ PULMONARY OLIGEMIA. (↓ Broncho vascular marking; due to ↓ CO due ↓ VR. ↓ filling)



2. Echocardiography: Good +ve & Good -ve

- The most sensitive test for detecting the presence of pericardial effusion & its severity.

3. Cardiac catheterization & angiocardiology: (not needed)

- Reveals a gap between the edge of the heart which contains the catheter and the peripheral cardiac shadow.
- Angiocardiology demonstrates the actual size of the heart inside the effusion.

III. OTHER INVESTIGATIONS FOR THE CAUSE:

[diagnose type of effusion then Cause]

other Causes

PERICARDIOCENTESIS:

- Pericardial biopsy:
- Serology: (antibodies)
- Urea & Creatinine:

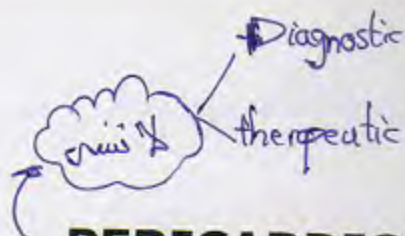
see later.

e.g. for TB or malignancy.
 for SLE.
 for Renal failure.

typical granuloma

More important to diagnose a bacteria e.g TB

miss staining
Culture



PERICARDIOCENTESIS:

= Tapping
(Aspiration of effusion)

- Indications of aspiration:

A] Diagnostic: "May be needed for diagnosis of the cause by analysis of the pericardial fluid"

a) Features of exudate & transudate effusions:

	TRANSUDATE	EXUDATE
① Proteins Fluid protein / serum protein	< 3 gm % < 0.5	> 3 gm % > 0.5 [↑ Fibrin, fibrinogens & Globulines]
② Specific gravity	< 1016	> 1016 (↑ Conc. of Ptn)
③ LDH Fluid LDH / serum LDH	< 200 IU / L < 0.6	> 200 IU / L > 0.6 [WBC release of LDH]
④ Cells (WBCs)	< 1000 / cmm	> 1000 / cmm

inflammatory fluid

esp. neutrophils

NB Characteristics of TB effusion: (exudate & special characters)

1. Exudate: rich in Lymphocytes & RBCs → hemorrhagic pericarditis.
(not neutrophils)
2. TB bacilli can be detected by:

- Staining: ZN stain.
- Culture: Lowenstein-Jensen medium or BACTEC.
- Animal Inoculation: Guinea pig.

• PCR

استن كثر

b) Features of Pyopericardium:

- The fluid is purulent & contains many pus cells.

c) Features of Hemopericardium:

- The fluid is bloody & contains many RBCs.

d) Features of Chylopericardium:

- The fluid is milky white & contains many fat, clears on addition of ether & stains orange with: Sudan III

B] Therapeutic:

(see later).

- in Cardiac tamponade: to relieve dyspnea & compression (symptomatic tt)
- in Malignant effusion: to drain pus & inject cytotoxic drug (palliative tt)
- in Suppurative effusion: to drain pus & inject antibiotics (curative tt)

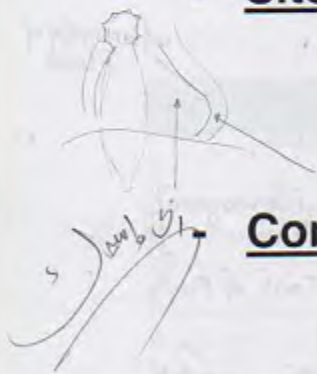
- Contraindications:

- Bleeding tendency.
- Uncooperative patient.

أضرار أخرى
Trauma
lung & pleura
pneumo-thorax
Rupture: myocardium

- Sites of aspiration:

- From outside the apex.
- From the bare area. (4,5th spaces ; 4cm lt to the sternum)



Complications of aspiration:

- Infection.
- Arrhythmias. (due to manipulation to heart)
- Trauma: myocardial rupture, pneumothorax.

DIFFERENTIAL DIAGNOSIS

1. Congestive heart failure:

- Dyspnea - Different types of dyspnea (exertional, orthopnea, PND).
- Ascitis - Oedema of LLs before ascites. (not ascitis precox.)
- Pulse - No pulsus paradoxus.
- Heart - Heart is enlarged & there may be murmur & gallop.

2. Constrictive pericarditis:

- Prominent y descent.
- Pericardial calcification: on Chest x-ray. تقشف أسرع
↳ on top of fibrosis.

• diastolic failure
• pulsus paradoxus

3. Restrictive cardiomyopathy.

4. Liver cirrhosis & nephrotic syndrome.

Generalized edema

5. DD of the cause of effusion.

Other causes of

فشل القلب

in Diagnosis Must be Pericardial effusion due to

TREATMENT

1. Treatment of the cause: e.g. TB:

- Anti - TB drugs.
- Corticosteroids: to ↓ inflammation & prevent constrictive pericarditis.

2. PERICARDIOCENTESIS: (Aspiration of effusion)

- In cardiac tamponade : *to relieve dyspnea & compression.* *therapeutic tapping*
- In suppurative effusion : *to drain pus & inject antibiotics.* *symptomatic relief.*
- In malignant effusion : *to inject cytotoxic drugs. (palliative)*
= leukemia *"antibiotic" استخدام الدواء مع تغيراته*

3. Pericardiodesis: (palliative) (Pericardial sclerosis with Tetracycline)

- In malignant effusion: *to prevent re-accumulation.* *(سده سده هييجل ثاني)*
(rapid accumulation after aspiration) فترات سعات

4. Pericardiectomy or pericardio-pleural window:

- For massive & recurrent effusion not responding to medical ttt.

Important points in Pericardial effusion

- ♥ TB is the most common cause.
- ♥ Prayer's position to relieve dyspnea.
- ♥ Pulsus paradoxus.
- ♥ Kussmaul's sign in neck veins.
- ♥ Echocardiography is the most important investigation.

CONSTRICTIVE PERICARDITIS

(Pick's disease)

ETIOLOGY

4 I

1. **I**diopathic.
2. **I**NFECTIONS:
 - Tuberculosis: the most common cause.
 - Bacterial, Viral, Fungal.
4. **I**mmunologic: RA (but not RF). "Rheumatoid arthritis"
5. **I**rradiation. (dry pericarditis $\xrightarrow{\text{قلب}}$ effusion $\xrightarrow{\text{قلبي}}$ Constrictive)
(exudate)

PATHOLOGY

inflammation : exudate

- Dense fibrous tissue adheres the 2 layers of the pericardium together and constricts the heart. Frequently it undergoes calcification.

PATHOPHYSIOLOGY

تسبب بحد من ال elasticity في الأول
"in late diastole" بحد من الطبيعي Limit ووصول

- The rigid pericardium interferes with ventricular filling in late diastole.
Ventricular filling is not reduced in early diastole, but it gets reduced abruptly when the elastic limit of the pericardium is reached.
This is in contrast to pericardial effusion where ventricular filling is reduced all through diastole.

CLINICAL PICTURE

Symptoms

1. Symptoms of: Systemic congestion.
2. Symptoms of: Low CO.
3. Symptoms of the cause: e.g. TB toxemia. night fever
loss of wt. night sweating
loss of appetite

No Pain : not fluid to stretch the parietal

No Pressure signs

Signs

1. Signs of the CAUSE.

e.g.

TB.

apical fibrosis
or
apical Cavitation

2. GENERAL signs:

I. Decubitus:

No special decubitus.

II. Signs of:

Systemic congestion:

a) Neck veins:

- Congested pulsating neck veins.

- Kussmaul's sign: (inspiratory filling)

Due to failure of the right side of the heart to accept the increased venous return during inspiration.

- Freidreich's sign: (diastolic collapse) "squeezed atrium by fibrosis"

Prominent y descent due to rapid emptying of the congested neck veins in a short time after opening of the tricuspid valve.

b) Enlarged tender liver.

c) Ascites before oedema of lower limbs

(ascites precox).

III. Signs of:

Low CO:

a) Low SBP, weak pulse, pallor.

b) PULSUS PARADOXUS:

in 30 % of the cases.

IV. AF:

(fibrosis around atrium → stretch)

in 30 % of the cases.

Not in Ventricle: thick Ms need severe fibrosis to be stretch: in v. severe late cases

3. CARDIAC signs:

A) Precordial examination:

- Apical pulsations:

weak or absent. (diminished motility) متعزيم

B) Auscultation:

- Muffled heart sounds.

• PERICARDIAL KNOCK:

- A sharp diastolic sound due to the sudden halting of the relaxing ventricles by the rigid pericardium.

INVESTIGATIONS

I. ECG:

- Low voltage. ($\downarrow$ motility)
- Diffuse nonspecific ST – T changes (Depressed ST, inverted or flat T).
- AF: in 30 % of the cases.

II. IMAGING:

1. CXR:

- Cardiac shadow is small.
- Calcification of the pericardium is diagnostic.
- Diminished cardiac pulsations under the screen.
- PULMONARY OLIGEMIA. ($\downarrow$ CO)

2. Echocardiography:

- Pericardial thickening. موجب آشوفها

3. Cardiac catheterization & angiocardiology:

- Square root sign:** "Early diastolic dip followed by a plateau" in:
RV & LV ventricular pressure tracings.

4. CT & MRI:

- Very sensitive in detecting: Pericardial thickening.

DIFFERENTIAL DIAGNOSIS

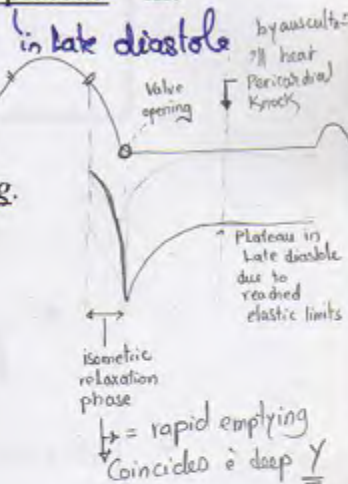
1. Congestive heart failure.

2. Pericardial effusion.

3. **RESTRICTIVE CARDIOMYOPATHY:** جدار قلب

4. Liver cirrhosis & nephrotic syndrome.

5. DD of the cause of constrictive pericarditis.



TREATMENT

1. Pericardiectomy:
 - Is the definitive therapy.
2. Treatment of the cause:
 - Anti – TB drugs (before & after pericardiectomy).

Important points in Constrictive Pericarditis

- ♥ TB is the most common cause.
- ♥ Pericardial knock.
- ♥ Calcification in chest x – ray.
- ♥ Restrictive cardiomyopathy is the main DD.
- ♥ CT or MRI is the most important investigation.

ADHESIVE PERICARDITIS

- * This condition may follow rheumatic fever which causes adhesions between the pericardium & the surrounding mediastinal structures & chest wall.
- * It has no significant hemodynamic effects & is rarely encountered.

Starts as infection → Antibody

- Glomerulonephritis
- Mycoplasma pneumonia → hemolytic Anemia
- ITP
- Guillain Barre
- DMT I

Treatment of the cause

Important points in Constrictive Pericarditis

1. TB is the most common cause.

2. Pericardial knock.

3. Calcification of chest X-ray.

4. Constrictive pericarditis is the main DD.

5. ECG or MRI is the most important investigation.

ADHESIVE PERICARDITIS

This condition may follow the acute stage of infection. Adhesions between the pericardium & the surrounding mediastinal structures develop.

It has no significant hemodynamic effect & is rarely symptomatic.

RHEUMATIC FEVER

- An inflammatory disease that occurs as a sequel to pharyngeal infections by group A hemolytic streptococci.
- It involves principally the: joint, heart, CNS, skin & subcutaneous tissue.

ETIOLOGY

Probably an autoimmune process mediated through either:

1. Antigenic similarity:

Antibodies formed against streptococcal antigens react also with human tissue antigens since both are immunologically identical.

2. Altered antigenicity:

Streptococcal antigens bind to human tissue antigens rendering them antigenic and thus stimulating antibody formation against them.

PREDISPOSING FACTORS

- [Age: Commonest between 5 & 15 years, Rare below 5 & above 25 years.
 - [Sex: Equal, except rheumatic chorea which is more common in females.
 - [FH: due to similar environment, (overcrowding & bad hygienic conditions).
 - [Recurrent streptococcal infections of the: pharynx & tonsils.
- Family History
Past History
- عشان كده بيتشيل الالوة

PATHOLOGY

"INFLAMMATION"

I. SITE:

1. Joints: synovial membrane. (arthritis)
2. Heart: pancarditis.
3. CNS. (Basal Ganglia) Chorea
4. Skin & subcutaneous tissue. (erythema marginatum & subcut. nodules)
5. Small blood vessels.
6. Serous membranes: pleura & peritoneum.

II. TYPES:

1. Exudative Lesion:

- Affects mainly: serous membranes.
- Resolves completely: without residual effect. *No fibrosis
No Chronicity*

2. Proliferative Lesion: “Aschoff nodule”:

- Affects mainly: the heart & the skin.
→ valve lesions. → nodules.
- Heals by fibrosis. *(leave residuals)*
- Consists of:
 - Central area of fibrinoid degeneration, *THEN:*
 - Macrophages, Lymphocytes & Aschoff's giant cells. *→ damage in Collagen fibers.*
 - Outer layer of fibroblasts. *→ fibrosis → Chronicity*



Similar Picture as TB
as Sarcoidosis

CLINICAL PICTURE

1- MAJOR CRITERIA

1. ARTHRITIS 75%. “more Common in adults”

- Polyarticular affecting large joints: e.g. knees, ankles, elbows.
- It is fleeting or migrating in character, leaving the affected joint free. HOWEVER, several joints may be affected simultaneously.
- The affected joint is painful, tender, red, hot, swollen with:
LIMITATION OF MOVEMENT. *(differentiate between major & minor Criterion)*
- It is more common in adults, while in children, carditis is more common.
- There is a characteristic response to Salicylates.
Arthritis → Asprine

Exudative lesion

- Site - Serous memb.
- Resolve Completely

Proliferative Lesion

- Heart, skin
- $\hat{=}$ residual Effect
- heal by fibrosis

NB

- Monoarthritis in metatarso phalangeal joint of Big toe $\rightarrow$ Gouty arthritis
- Metacarpophalangeal joint of patient aged 20-40y $\rightarrow$ rheumatoid arthritis

Exudative lesion

Exudative lesion

Heart, skin
residual effect
local by factors

Exudative lesion
Residual (highly)
of factors

II. TYPES:

1. Exudative

2. Proliferative

NE

Monocytes in nodules phagocytose point of pig fat - Gouty arthritis
late capillary point of effort of fat - rheumatoid arthritis

1. ARTHRITIS

(1) new lesion

NEB Arthritis is induced by : ① Infection
③ Dilatation

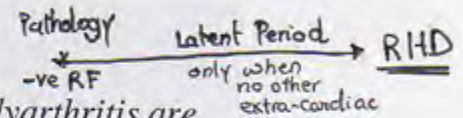
② Injury & Manipulation
④ Ischemia

2. CARDITIS 40~50% "More common in Children"

Children
Cortisol

- It could be a pancarditis.

- It could be asymptomatic, Therefore:



Unless extra-cardiac manifestations as polyarthritides are associated, the condition will be missed. Then, later in life RHD may be recognized without definite history of RF. (Latent period)

- Rheumatic carditis may present with:

I. Endocarditis: (Valve) → Commonly Lt side

1. Mitral Valvulitis: (More Common)

a) Mitral Regurge. — early stage of acute inflam. → damage the valve
stage of Fibrosis → retraction of cusps.

b) Mitral Stenosis: (More Common)

early acute stage of inflammation

- Carey Coomb's murmur: mid-diastolic murmur develops early in the acute stage due to swelling of the mitral cusps causing narrowing of the mitral orifice.

late stage of fibrosis

- Murmur of organic mitral stenosis: later on fibrosis occurs leading to permanent organic mitral stenosis, long after subsidence of the acute stage.

2. Aortic Valvulitis:

a) Aortic Regurge.

b) Aortic Stenosis.

II. Myocarditis: Global ↓ in Contractility → impending HF

Normally each degree elevation

↑ HR by 10 beats/min

37 → 40 : 90 → 120

1. Tachycardia disproportionate to the degree of fever. due to added effect of compensatory mech. to HF

2. Tic-Tac rhythm:

S_1 & S_2 are similar due to loss of the muscular component of S_1 .
(which is already absent in S_2)

3. Cardiac enlargement & may be heart failure. (try to compensate HF)

4. Cardiac arrhythmias & may be heart block.

inflammation → excitability
↳ Block AV Bundle

5. Gallop rhythm. (weak Myocardium; S_3 & tachy) Flappy = weak

6. Functional murmurs: due to ventricular dilatation. (try to compensate HF)
(Mitral & Tricuspid regurge)

III. Pericarditis:

• May be dry or with effusion. (exudate)

• No constrictive pericarditis in rheumatic fever. (Never fibrosis in Serous membrane)

• Exudative lesion.

Remember: Relative Bradycardia in GIT

3. CHOREA (Sydenham's chorea) * 8-10%

- Rapid involuntary movements (details in neurology). *pseudoparaparesis*
- Never associated with arthritis.
- More common in females.

subcut. nodules in RF is MUCH LESS common than rheumatoid arthritis.

4. SUBCUTANEOUS NODULES (rare)

- Painless nontender small swellings:
 - a) Over bony prominences & tendons, e.g. dorsum of hands & feet. (*extensor*)
 - b) Not adherent to the skin.
 - c) Painless not tender

5. ERYTHEMA MARGINATUM (rare)



- Painless nontender erythematous (red) spots:
 - a) On the trunk & proximal extremities in crops. (*multiple*)
 - b) The margin is serpiginous.
 - c) Gradually enlarge while the centers become pale, then: they disappear & reappear. [*in crops again*]



2. MINOR CRITERIA 1 + 2 + 2

1. Previous rheumatic fever.

infect 2. Present fever.

3. Arthralgia.

[*العيان هيبة يا لما Arthralgia يا لما arthritis*]

infect 4. Acute phase reactants: leukocytosis, ↑ ESR, ↑ CRP.

5. PR interval in ECG:

= AV Conduction
1st° AV Block

Prolonged.

> 0.2 sec
> 5 small squares



3. OTHER MANIFESTATIONS

Pleura 1. Pleurisy. (auto antibody reaction)

2. Pneumonia. (non-infective) (antibodies ...→ like that in SLE)

Peritoneum 3. Pain in the abdomen simulating acute abdomen. (Non-infective peritonitis auto antibody reaction)

4. Erythema nodosum. (painful)

* The British physician Thomas Sydenham (1624-1689), described chorea.

* Some Medical Causes of Acute Abdomen :

- ① DKA
- ② Inferior Myocardial infarction
- ③ Basal Pneumonia
- ④ Familial Mediterranean fever
- ⑤ Hemolytic Crisis

105

Erythema marginatum



Erythema nodosum

Red painful nodule on the skin overlying shin of Tibia

- Important question: "Self – assessment"

"Mention other causes of erythema nodosum." 35 T

- ① - Post Streptococcal infection
- 35 ② - Sulphonamide S.E. (Allergy)
- ③ - Sarcoidosis
- T ④ - TB

105

WhiteKnightLove

INVESTIGATIONS

A] Cardiac investigations:

1. ECG: cardiac enlargement, prolonged PR interval.
2. CXR: cardiac enlargement.
3. Echocardiography: cardiac enlargement, valve lesions.

B] Laboratory investigations: 3+2

1. Complete blood picture: anemia & leukocytosis.
2. C reactive protein (CRP): positive.
3. ESR: elevated.

* Anemia due to:
 ① toxic inhibition BM
 ② ACD
 "Anemia of chronic disease"
 Chronic infection e.g. TB → chronic inflammation e.g. SLE → Malignancy

4. Streptococcal antibody tests:

a) Anti-streptolysin O titer (ASO)

- Titer higher than 250 todd units in adults & 400 todd units in children is indicative of recent streptococcal infection.
- Rising titer is more diagnostic.

b) Anti-streptozyme test (ASZ)

- Titer higher than 200 units / ml is present in nearly all patients with acute rheumatic fever.

5. Throat culture:

- May reveal group A streptococci.
- Less satisfactory than streptococcal antibody tests in diagnosing recent streptococcal infection. (as disease is Post-streptococcal)

DIAGNOSIS OF RHEUMATIC FEVER

"Revised Jones Criteria"

- The diagnosis is established by the presence of **2 points**:

1. Two major criteria or one major & 2 minor criteria.

② Evidence of recent streptococcal infection:

"Positive streptococcal antibody tests, or Positive throat culture".

* Evidence of recent streptococcal infection.

↑ ESR: ① ^{acute}Severe infection (eg TB)

② ^{acute}Severe inflammation (eg autoimmune diseases; active Rheumatoid & active SLE)

③ Malignancies

④ Anemia

↓ ESR: ① Cryoglobulinemia

② polycythemia

Sam
D.D:RF ① Infective Endocarditis (fever; joint & heart)

DIFFERENTIAL DIAGNOSIS

- ① **INFECTIVE ENDOCARDITIS:** تآكل السطح
- ② Causes of: fever in a cardiac patient.
- ③ Other causes of: acute arthritis, carditis, chorea.
- ④ Precipitating factors of: heart failure. [e.g infections ←]

TREATMENT

A] Prophylactic

1. Primary (to prevent rheumatic fever)
 - Proper management of pharyngeal infections.
 - Tonsillectomy for chronically infected tonsils.
2. Secondary (to prevent recurrence of rheumatic fever; rheumatic activity)

- **Long acting penicillin:** (Benzathine penicillin G)

1.2 million units given monthly IM until the age of 25, or for 5 years after the last attack (whichever is longer), or for life. أبداً بعد

- **Erythromycin:** (for patients allergic to penicillin)

250 mg / 12 h orally.

NOT
Curative

B] Therapeutic

(only supportive) (Not Curative)

- There is no specific treatment for RF & none of the measures will change the course of the attack. However, good supportive therapy can reduce mortality & morbidity.

1. Rest: until signs of inflammation disappear.

2. Diet: light, poor in salt.

Myocardium خفيف

3. Antibiotics: (for eradication of streptococcal infection) ^{اللي موجود دلوقتى}

- **Benzathine penicillin G**: (Single IM injection)
1.2 million units (600.000 units for children below 10 years).
- **Erythromycin**: (for patients allergic to penicillin)
250 mg / 6 h orally for 10 days.

4. Anti-inflammatory drugs:

- These drugs relieve the manifestations of RF, BUT:
do not prevent chronic valvular lesions.

a) **Aspirin** (Acetyl salicylic acid): For Arthritis:

- 100 mg / kg / day orally in 6 divided doses until there is a satisfactory response (6 weeks).

- Antacids should be given to prevent gastritis. <sup>وينا خذ على صومه حلياً
بعد عبات كبر</sup>

b) **Corticosteroids**: For Carditis:

- **Prednisone**:

1 mg / kg / day orally in 4 divided doses for 4 weeks,
then gradual withdrawal in 4 weeks. (to prevent acute adrenal failure)
(Addisonian Crises)

- **Aspirin**:

is given during withdrawal of steroids & continued for additional 4 weeks.

^{"Ant-pituitary" $\leftrightarrow$ "Ant-Adrenal"}
<sup>مع اني كره كره
مبعضه تشين مع ال
withdrawal.</sup> **ACTH**: [to stimulate suprarenal]
1 ampoule IM twice / week in the last week <sup>of Prednisone tit
of withdrawal</sup>

5. Treatment of chorea: (Refer to neurology).

The most important points in Rheumatic fever

- ♥ Autoimmune etiology.
- ♥ The most common cause of valvular heart diseases, especially MS.
- ♥ Diagnosed according to: "Revised Jones criteria".
- ♥ Investigations have a minor role in diagnosis compared to clinical picture.
- ♥ Treated by Aspirin for Arthritis, & by Corticosteroids for Carditis.

	1st w	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th	12th
Corticosteroids												
Aspirin					✓	✓	✓	✓	✓	✓	✓	✓
ACTH								● ●				

Drugs Must be Gradually withdrawn or all die :

- ① Corticosteroids
- ② Anti-epileptics (status epilepticus)
- ③ Anti-coagulants ! (Rebound thrombosis)

Uses of Cortison in Cardiology

- Carditis of RF
- Non Cardiogenic pulm.-edema
- Angioneurotic
- Post Cardiotomy syndrome

Dose do not change
 Rh. arthritis
 --- anemia

- **Important question:** **"Self – assessment"**

"Mention important post – streptococcal sequelae."

- Henoch Schonlein
- GN

- **Important question:** **"Self – assessment"**

"Mention skin manifestations of rheumatic fever."

- 2 Major : - Erythema marginatum
- Skin nodules
- 1 Other : - Erythema nodosum

ENDOCARDITIS

DEFINITION

- Inflammation of the endocardial surface of the heart.

I. INFECTIVE ENDOCARDITIS:

1. According to the causative organism:

- Bacterial endocarditis.
- Non – bacterial endocarditis. (Fungal) (Viral)

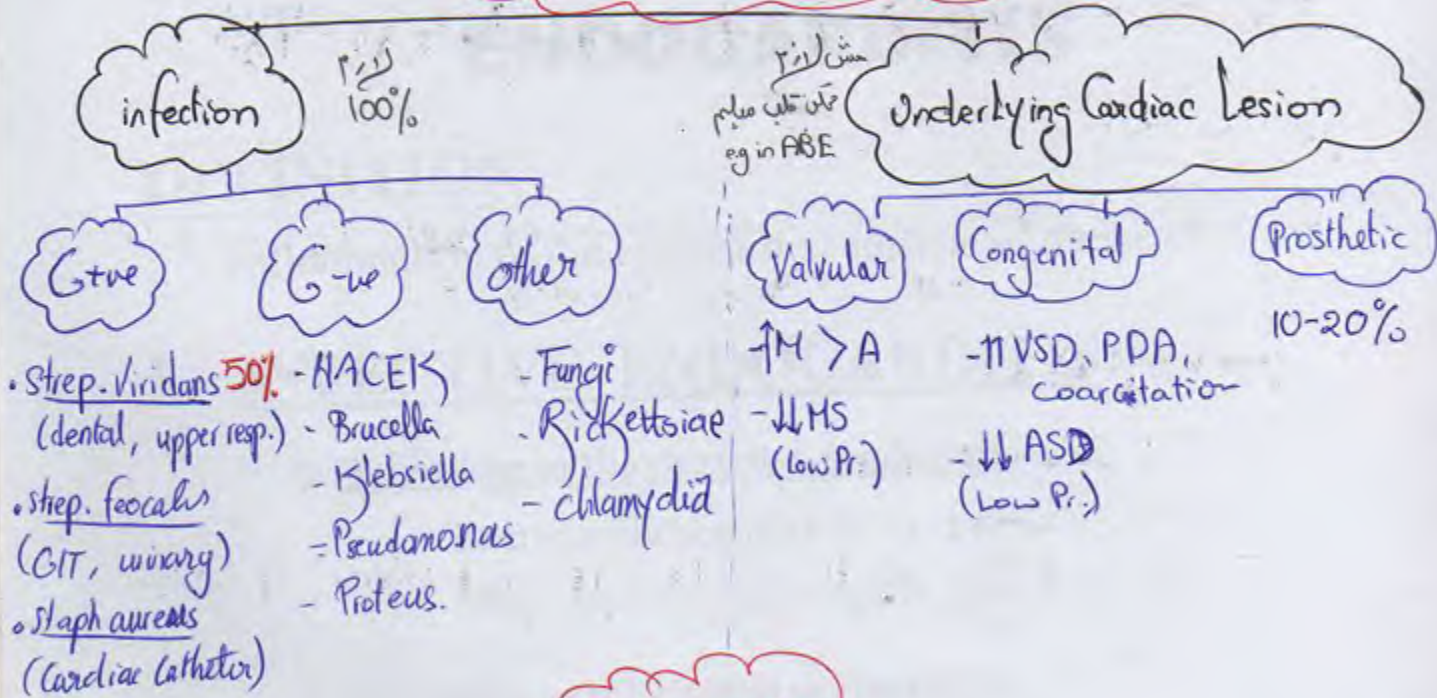
2. According to the clinical presentation:

- Subacute bacterial endocarditis (SBE):
 - Onset: Insidious.
 - Heart: Diseased heart.
 - Organism: Low virulence, e.g. Strept. viridans.
 - Prognosis: Low mortality.
 - Incidence: Common.
- Acute bacterial endocarditis (ABE):
 - Onset: Acute.
 - Heart: Diseased heart or normal heart.
 - Organism: High virulence, e.g. Staph. aureus.
 - Prognosis: High mortality.
 - Incidence: Rare.

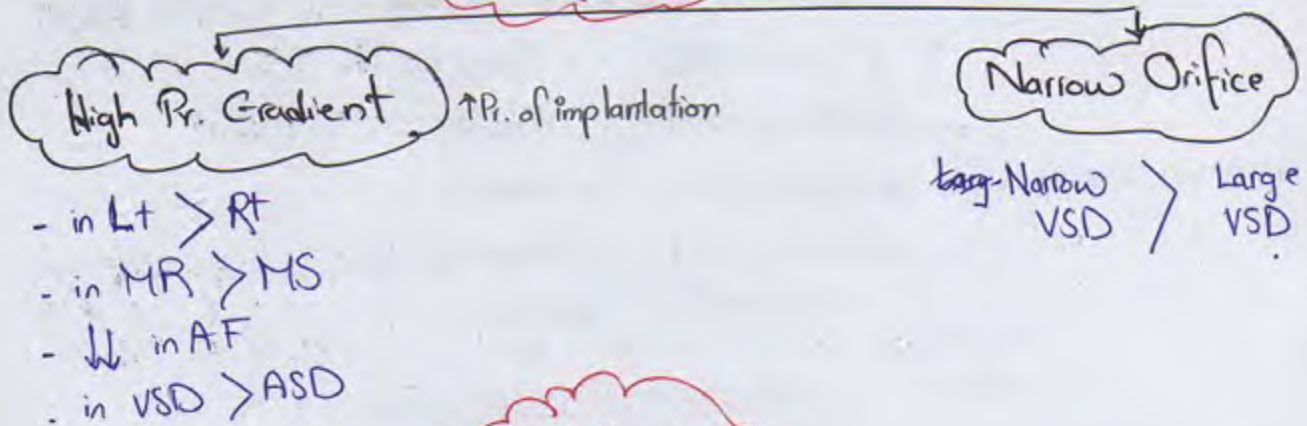
II. NON – INFECTIVE ENDOCARDITIS:

e.g. Rheumatic fever, SLE. "autoimmune disease"

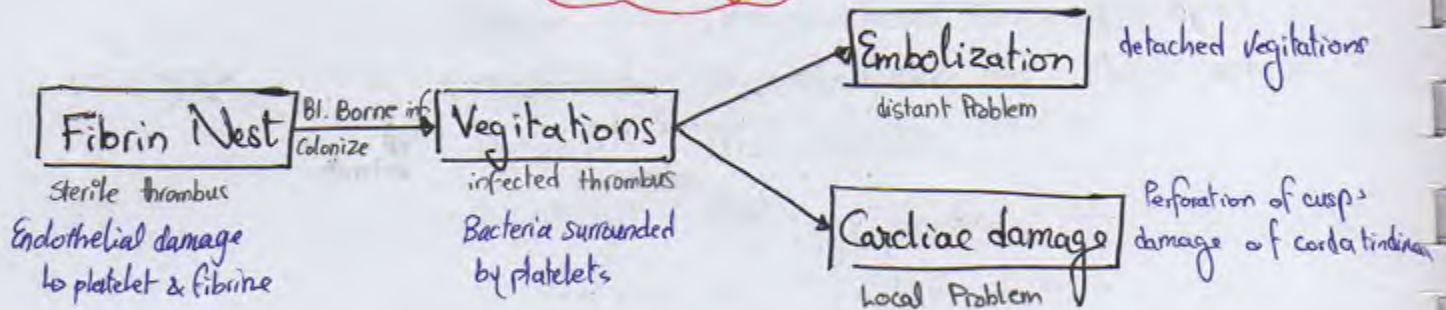
Etiology Infective endocarditis



Pathogenesis



Pathology



Manifestations are due to

- Toxemia
- Embolization
- Cardiac damage
- Immune Complexes

INFECTIVE ENDOCARDITIS

ETIOLOGY

long Q + P₁₂ + I

- For infective endocarditis to develop, there should be a combination of 2 factors:

Infection + underlying cardiac lesion.

I. INFECTION:

a) The causative organism:

- Gram-positive cocci:
 - Strept. viridans (the most common organism) "40 - 50 %".
 - Strept. foecalis & Staph. aureus.
- Gram-negative bacilli:
 - HACEK group:
 - Hemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella.
 - Klebsiella, Brucella, Pseudomonas, Proteus.
- Other organisms: Fungi, Rickettsiae, Chlamydia.

b) The route of infection:

- Bacteremia
- Dental procedures & upper resp. surgery: (usually Strept. viridans).
 - GIT & Genitourinary procedures: (usually Strept. foecalis).
 - Cardiac surgery & Catheterization: (usually Staph. aureus).

(inoculation from skin)

II. UNDERLYING CARDIAC LESION:

a) Valvular lesions:

- The mitral valve is most commonly affected followed by the aortic valve.
- The disease is not common in MS due to:
 - Low pressure gradient.

b) Congenital anomalies:

- Especially VSD, PDA & coarctation.
- The disease is not common in ASD due to:
 - Low pressure gradient.

c) Prosthetic valves: accounts for 10 - 20 % of the cases.

rough surface "deposits infected thrombi"

More rough exposed surface
 injury الى ما يكون
 More rough exposed surface

PATHOGENESIS

- Two factors are needed to increase the incidence of infective endocarditis:

I. High pressure gradient: (Pr. of implantation)

- It is common on mitral & aortic valves (valves of the left side).
- It is rare on tricuspid & pulmonary valves (valves of the right side).
- It is more common in MR than in MS.
- It is more common in VSD than in ASD.
- It is RARE in: AF. (eg in MS)

II. Narrow orifice:

- It is more common in small VSD than in big VSD.

PATHOLOGY

Fibrin nests: = sterile thrombus

Endothelial damage leads to deposition of platelets & fibrin (fibrin nests).

Vegetations: = infected thrombus.

Blood-borne organisms colonise these nests, forming vegetations.

Therefore: vegetations are composed of bacteria surrounded by platelets & fibrin.

Embolization: (distant problem)

Vegetations may detach leading to embolization

(septic emboli)
 Lt → system
 Rt → pulm.

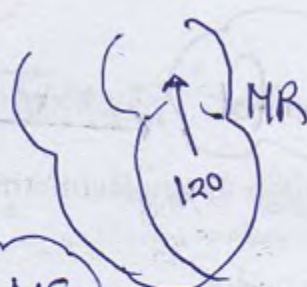
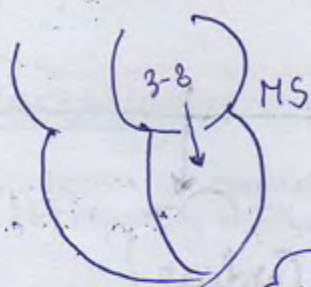
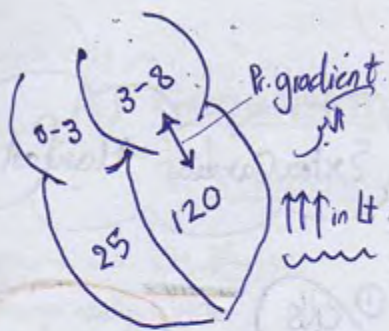
Cardiac damage: (local problem)

Infection may result in damage & perforation of the cusps & chordae tendinae.

CLINICAL PICTURE

- **Manifestations of the disease are due to:**

1. Toxemia.
2. Cardiac damage.
3. Embolization.
4. Immune complexes. "GN"



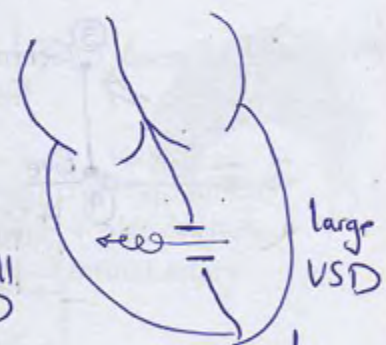
MR > MS



AF = Ms
No contraction
No force to pass bl.
↓ Pr. of implantation



Pass harder
↑ Pr. of implantation



Pass easily
↓ Pr. of implantation

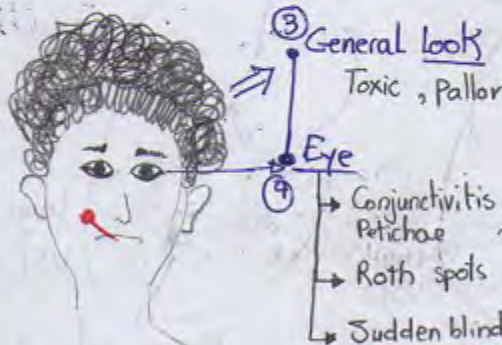
General Examination

C/P infective Endocarditis

5+3+5

General Manifest

- ① T: Fever, resistant to Antipyretics + Headach, malaise
- ② Pulse: Tachy Cardia
May be absent in some (embolization)

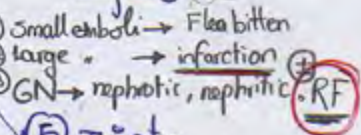
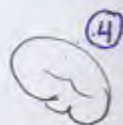
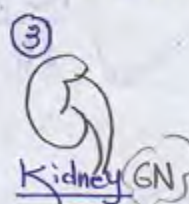
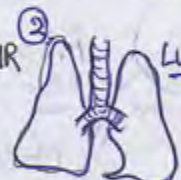


Cardiac

- ① Lesion
- ② Murmurs
change new Sea Gull
- ③ ± HF
- acute HF in acute HR
- ↑ BMR
- damaged Myo.

Other Systems Review

Extra Cardiac Manifest



⑤ Hands & L.L.

- ① Pale clubbing
- ② Splinter Hge
- ③ Osler's nodules
- ④ Janeway lesions

	Toxemia	Emboli	Immune Complexes	Cardiac damage
<u>General</u>				
- T, General look	✓			
- Eye	• Roth Spots (oval Hge) • Petichae (conjunctiva)	• Sudden Blindness • Petichae (Conjunctiva) • absent Pulse • splinter Hge	• Roth spots (pale center) • osler's nodules • Janeway lesions	
- Hand, UL, UL	• clubbing • splinter Hge			
<u>Cardiac</u>				✓
<u>extra Cardiac</u>				
- CNS		• stroke (Hemiplegia) • SAH "rupture Myc. aneurysm"		
- Lung		• Pneumonia • embolism		
- Kidney		• Flea bitten } hematuria • infarction	• GN → nephrotic, nephritic → RF	
- Spleen	• Slightly soft enlarged tender	• splenic infarction • rub • perisplenitis	• slightly enlarged, soft & tender	
- Joints			• arthritis, arthralgia	

5 A) General manifestations: of "Toxemia" + emboli

1. Temperature: Fever & general constitutional manifestations.

"Not responding to Antipyretics"

2. Pulse:

- Tachycardia. ^{peripheral}
- May be absent pulsations due to embolization.

3. General look: Marked pallor & toxic facies.

"toxic inhibition to BM" (No Anemia in Acute disease)

4. Eyes:

- In the conjunctiva: Petichae (due to microemboli & toxic capillaritis).
- In the retina: Roth spots (oval hemorrhages + pale center).
- SUDDEN BLINDNESS: due to embolism of the central retinal artery.

(Hge)
toxic capillaritis



infarcted area

(immune-complex
vasculitis)

5. Hands & lower limbs:

- Pale clubbing. toxic
- Osler's nodes: ¹ (immune complexes)
Small painful red tender raised, in the pulps of the fingers & toes.
They are due to: immune hyperplasia of the capillary endothelium.
- Janeway's lesions: ²
Small painless red-blue nontender macules, in the palms & soles.
- Splinter hemorrhages:
Linear hemorrhages under the nails (due to microemboli & toxic capillaritis).

3 B) Cardiac manifestations: (Localizing Manifestations)

1. UNDERLYING CARDIAC LESION.

2. MURMURS:

- o Change in the character of the already present murmurs.
- o Development of new murmurs.
- o Rupture cusps may give rise to a loud musical murmur:

"SEA GULL MURMUR" (Musical)

May lead to
Acute HF
due to Acute MR

3. HEART FAILURE

due to Metabolism
Local Cardiac damage

¹ Sir William Osler (1849-1919), Canadian, had called attention to their diagnostic importance.

² The American physician Edward G. Janeway (1841-1911), described the lesions.

C) Extra-cardiac manifestations:

Embolization

Immune Complexes (I.C.)

Circulating -
[Ag + Ab + Complement]

1. Spleen:

↑ RES Hyperplasia

- o Slightly enlarged soft & tender.

in Rt Hypochondrium

(inflamm. in capsule)

Stitching pain & splenic rub may occur due to embolization causing splenic infarction & perisplenitis.

2. Kidneys:



- o Renal infarction: (Big embolus) causing gross hematuria.
- o Flea-bitten kidney: (small emboli) causing microscopic hematuria.
- o Acute glomerulonephritis immune - complex GN:
 - Nephritic syndrome or nephrotic syndrome.
 - Renal failure.

3. CNS:

Commonest Presentation ←

الأشهر

- o Embolic hemiplegia.

"STROKE"

septic

microembolic to vessels of Vasa Vasorum

↓ weakens wall (infection)

- o SAH:

(Sub arachnoid Hge)

due to rupture of mycotic cerebral aneurysm.
= intracranial Hge → stroke

4. Lungs:

[Septic infected embolus]

- o Pulmonary embolism or pneumonia in cases of:
right-sided endocarditis (uncommon).

5. Joints:

Immune Complexes →

- o Arthralgia or Arthritis.

COMPLICATIONS

= Causes of Death

1. Heart failure.

2. Renal failure.

3. Embolization.

4. Rupture mycotic aneurysm.

(Bleed to Death)

5. Complications of treatment.

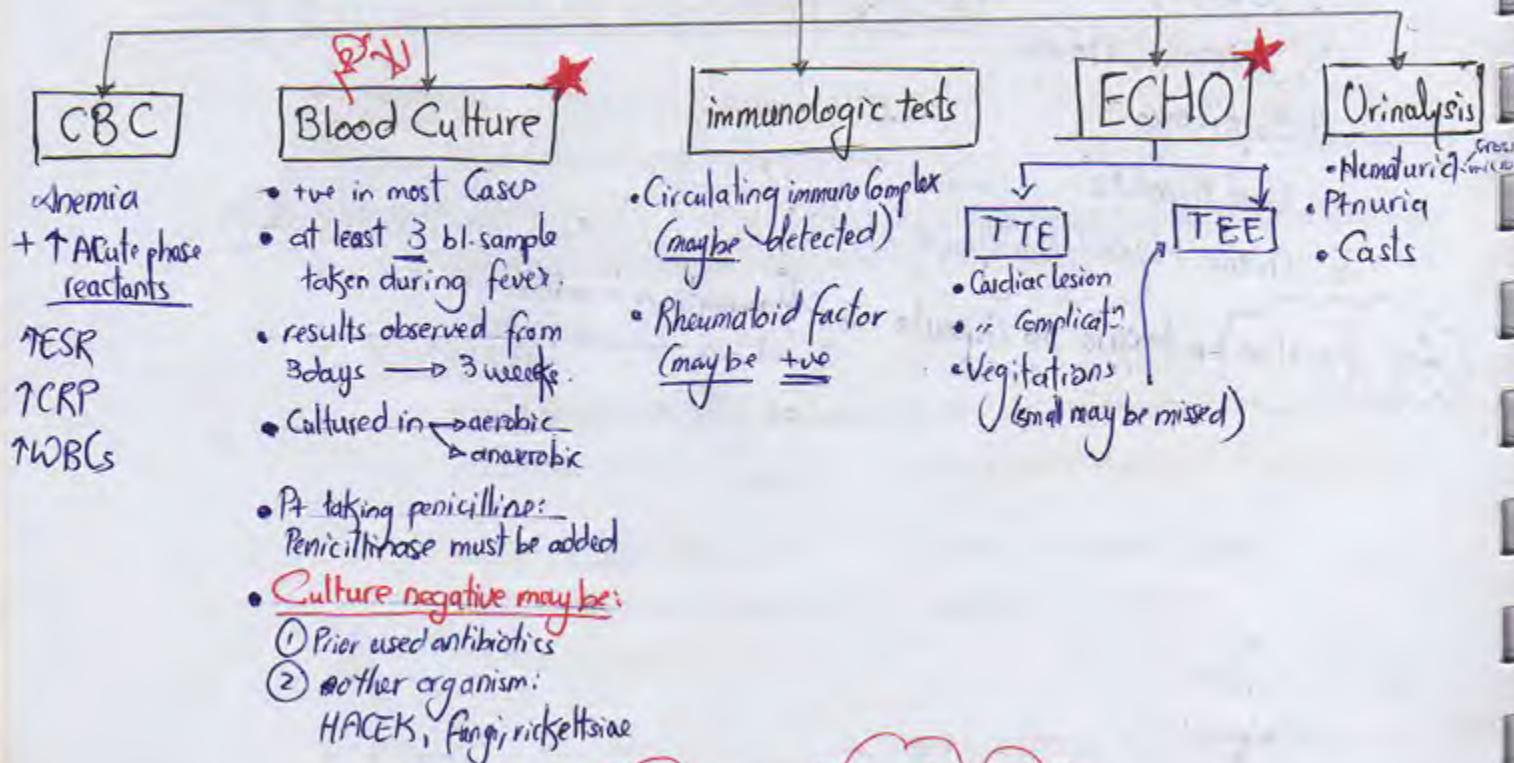


Enlarged tender Spleen

- 1- Infective endocarditis (~~decker~~)
- 2- Other infection (typhoid, brucella, infectious mononucleosis, Malaria)
- 3- Splenic Abscess
- 4- Septicemia
- 5- Viral Hepatitis
- 6- Chronic Myeloid Leukemia

Any infarction → toxins to Capsule → inflammation → Pain
Friction → Rub

Investigations for I.E.



Criteria for diagnosis for I.E.

Modified Duke Criteria

Major Criteria

- ① +ve blood Culture
2 separate blood culture to be +ve in atypical organism as streptococci
- ② +ve ECHO finding:
e.g. Vegetations
- ③ New Valvular Regurgitation:
- change of character of old murmurs
- Development of new murmurs
- Sea Gull murmurs

Culture

ECHO

Cardiac Damage

Minor Criteria

- ① Predisposition:
e.g. IV heroin addict
- ② Fever $\geq 38^\circ\text{C}$
- ③ immunological phenomena:
- GN
- Roth spots
- Osler's nodules
- ④ Vascular phenomena:
- stroke, intracranial Hge
- splinter Hge
- arterial emboli
- ⑤ +ve blood culture
without meeting major criteria (e.g. +ve Once.)

3P form

Toxemia

Immune complex

Emboli

Culture

Diagnosis: Clinical Criteria for Definit I.E. include

- 2 major OR
- 1 major + 3 minor OR
- 5 minor

SPECIAL FORMS



• Endocarditis in IV drug abusers:

needle sticks
skin

- It may occur on a normal heart.
- Staph. aureus is most common & TV is most commonly affected. ^{+ aortic valve}
- Pulmonary embolism & pneumonia are common.

• Fungal endocarditis:

- Often due to: Candida or Aspergillus.
- Of bad prognosis. ^{immuno-compromized}
- Associated with negative blood culture.

INVESTIGATIONS

1. Blood picture:

- Anemia. (toxic inhibition to BM.)
- Leucocytosis
- ↑ ESR
- ↑ CRP



Acute phase reactants

2. BLOOD CULTURE:

"the most important investigation"

Bacteremia

- It is positive in most of the cases. (Not in All)
- At least 3 blood samples are taken during fever.
- The results are observed from 3 days up to 3 weeks.
- Samples are cultured under aerobic & anerobic conditions.
- If the patient was taking penicillin, penicillinase enzyme should be added.
- Culture-negative endocarditis may be due to:
 - Prior use of antibiotics. (e.g. penicillins)
 - Infection with other organisms, e.g.: hacek, fungi, rickettsiae.

3. ECHOCARDIOGRAPHY:

a) Trans-thoracic echo:

- ①. The underlying cardiac lesion.
- ②. Any heart complication.
- ③. VEGETATIONS, however, small vegetations may be missed.

Invasive?
More accurate

b) Trans-oesophageal echo: “the most important investigation” TEE

- Detects SMALL VEGETATIONS missed by trans-thoracic echo.

أسرع في الكشف - لا حاجة لـ TTE
Blood culture
المرضى

4. Urine analysis: “GN”

- Hematuria: Gross or microscopic.
- Proteinuria: and casts.

5. Immunologic tests:

- Circulating immune complexes: may be detected.
- Rheumatoid factor: may be positive.

[(V. non specific) : Ab present in infective endocarditis]

6-2

DIFFERENTIAL DIAGNOSIS

- ◀ ——— ○ RHEUMATIC FEVER.
- heart ○ Causes of: fever in a cardiac patient.
- Brain ○ Causes of: embolization. (Neuro)
- Kidney ○ Causes of: GN. (Kidney)
- spleen ○ Causes of: tender splenomegaly.
- ◀ ——— ○ Precipitating factors of: heart failure.

DIAGNOSIS OF INFECTIVE ENDOCARDITIS

[MODIFIED DUKE CRITERIA]

③ Major criteria

[Confirmatory sign]

- invest. 2 ↑ 1
1. Positive blood cultures (2 separate cultures for a typical endocarditis microorganism, such as Streptococcus viridans).
 2. Positive echocardiographic findings: e.g. VEGETATIONS.
 3. New valvular regurgitation:
 - Change o Change in the character of the already present murmurs.
 - New o Development of new murmurs. (including Sea Gul Murmur)
4. Lowly Murmurs
Cardiac damage

⑤ Minor criteria

1. Predisposition (history of IV drug use or congenital heart disease).
- Toxemia 2. Fever $\geq 38^{\circ}\text{C}$.
- Emboli 3. Vascular phenomena (e.g. arterial emboli, intracranial hemorrhage, conjunctival hemorrhage).
- Immune Complexes 4. Immunologic phenomena (e.g. GN, Osler nodes, Roth spots).
5. Positive blood cultures: without meeting the criteria above (major criteria).
 - +ve Once
 - +ve for uncommon organism.

Clinical criteria for definite infective endocarditis include:

- 2 majoror,
- 1 major and 3 minoror,
- 5 minor criteria.

TREATMENT

I. Prophylactic:

1. Correction of the underlying cardiac lesion.

2. Prophylactic antibiotics:

- For dental procedures or upper respiratory surgery:

- *Amoxycillin*: 3 gm orally, 1 hour before the procedure, and, 1.5 gm orally, 6 hours after the procedure.

- For GIT or genitourinary procedures:

- *Ampicillin*: 2 gm IV **plus** *Gentamycin*: 80 mg IV, ½ hour before the procedure, and, 6 hours after the procedure.

- For cardiac surgery or catheterization:

- *Cefotaxime*: 2 gm IV, 2 hours before the procedure, and, 1 gm / 6h for 4 doses after the procedure.

II. Therapeutic:

A) MEDICAL TREATMENT:

1. Specific treatment: “Antibiotics”

- Once infective endocarditis is suspected, ttt should be started immediately without waiting the result of blood culture.
- TTT should be continued for at least 4 – 6 weeks.

a) For Streptococci:

- Penicillin G 20 – 30 million units / day IV for 4 weeks **plus**
- Gentamycin 1 mg / kg every 8 hours IV for 4 weeks.

NB For penicillin allergy, penicillin is replaced by Vancomycin 15 mg / kg every 12 hours IV.

b) For Staphylococci & gram-negative bacilli:

- Cloxacillin 2 gm / 4 hours IV *plus*
- Gentamycin 1 mg / kg every 8 hours IV.

c) For fungal endocarditis:

- Amphotericin B.

2. Symptomatic treatment:

- a) Rest: until clinical manifestations improve.
- b) Diet: light, poor in salt.
- c) TTT of complications.

B) SURGICAL TREATMENT:

- Replacement of the severely damaged valve.
- Replacement of the infected prosthesis.

ANATOMY OF THE CORONARIES

CORONARY ARTERIES

- There are **two main** coronary arteries which originate from the root of the ascending aorta:

1. The left coronary artery:

- It arises from the left sinus of Valsalva, passes forwards and to the left in the left atrioventricular groove for a short distance and then divides into **2 branches**:

a) The anterior descending artery:

It passes downwards in the anterior interventricular groove to the apex and then turns backwards to meet the posterior descending artery.

b) The circumflex artery:

It continues its course in the atrioventricular groove to meet the right coronary.

2. The right coronary artery:

- It arises from the right sinus of Valsalva, and then runs in the right atrio-ventricular groove to the posterior surface of the heart to meet the circumflex artery.

In the back of the heart it gives the posterior descending artery which runs downward in the posterior interventricular groove to meet the anterior descending artery.

PATTERNS OF CORONARY SUPPLY

1) Balanced circulation:

- The left coronary artery supplies:
The LA, LV and the anterior part of the interventricular septum.
- The right coronary supplies:
The RA, RV and the posterior part of the interventricular septum.

2) Right coronary predominance:

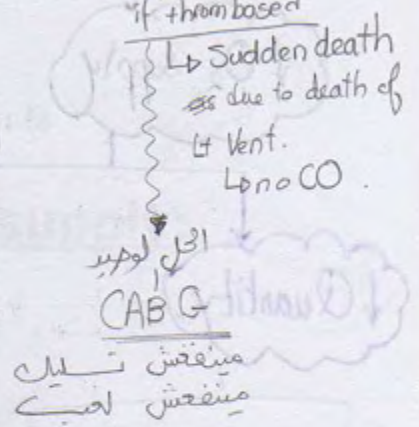
- The right coronary supplies also: *The posterior wall of the left ventricle.*

3) Left coronary predominance:

- The left coronary supplies also:
The posterior wall of the right ventricle & The posterior part of the septum.

Anatomical
Variation but
Still normal

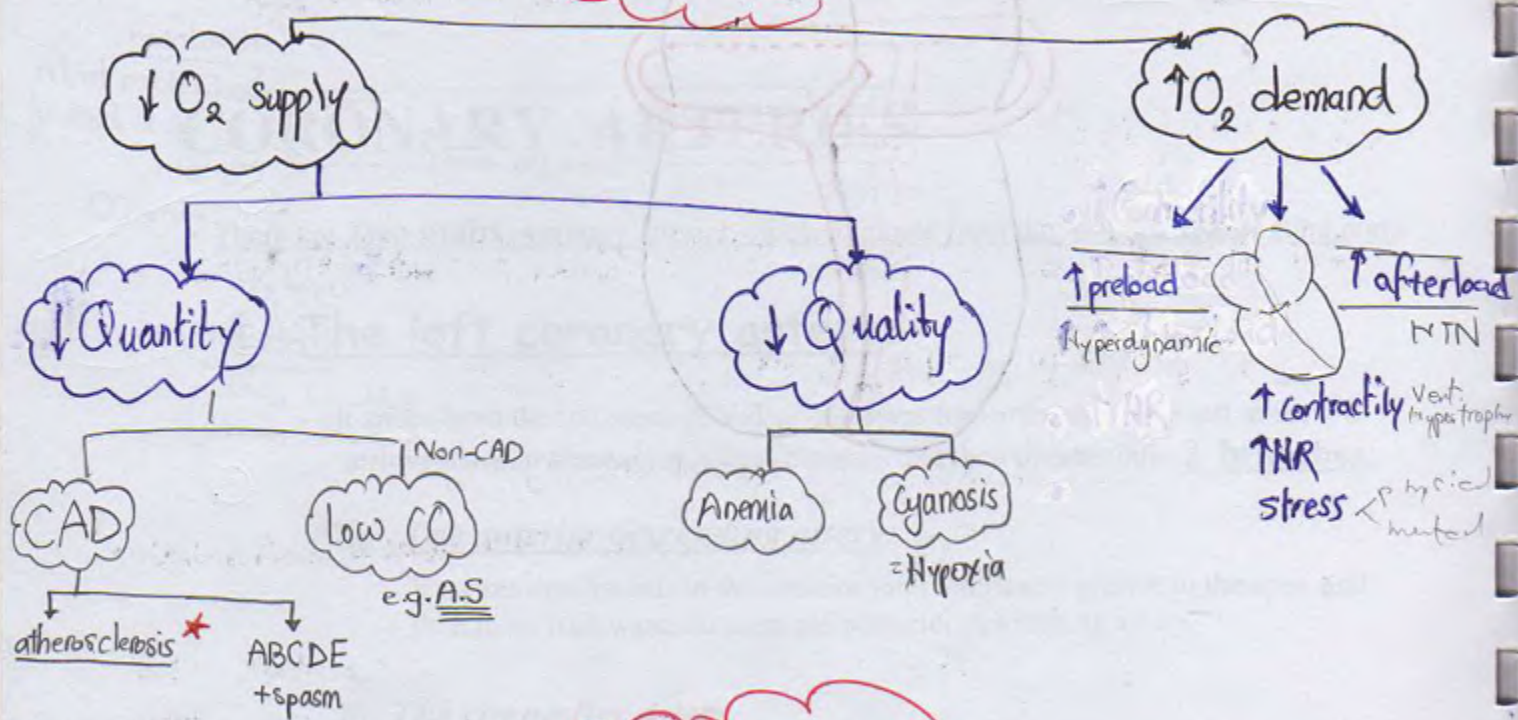
3-4 cm
LH main coronary artery



مِنْغُشْ لِيلِ
مِنْغُشْ لَحَبِ

Coronary a.

IHD Etiology



Atherosclerosis

2. The right coronary artery:

PATTERNS OF CORONARY SUPPLY

1) Right coronary artery

The right coronary artery (RCA) is the most common pattern of coronary supply, accounting for 40-50% of the population. It supplies the right ventricle and the posterior part of the left ventricle. The RCA is usually the largest of the three coronary arteries.

2) Left coronary artery

The left coronary artery (LCA) is the second most common pattern of coronary supply, accounting for 30-40% of the population. It supplies the anterior part of the left ventricle and the anterior part of the right ventricle.

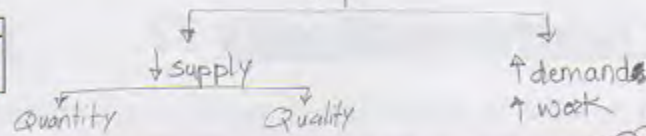
3) Left coronary artery

The left coronary artery (LCA) is the third most common pattern of coronary supply, accounting for 10-20% of the population. It supplies the anterior part of the left ventricle and the anterior part of the right ventricle.

ISCHEMIC HEART DISEASE

insufficient Blood supply

ETIOLOGY



مش بالضرورة يبقى
Coronary
يعنى CAD
IHD جرس

1) Decreased myocardial oxygen supply:

a) Decreased quantity of coronary blood:

1. CORONARY ARTERY DISEASE: (CAD)

99% لو حصة عامل

i) CORONARY ATHEROSCLEROSIS (most common cause).

ii) OTHERS:

- **Arteritis**, PAN = Poly arteritis nodosa, antibodies to arteries → endarteritis obliterans e.g. PAN, SLE. disease of small, medium sized not a disease of big vessels
- **Blood diseases, (Hypercoagulable)** e.g. PRV, DIC. + Contraceptive Pills. Polythemia Rubra Vera Hyper viscosity
- **Congenital anomalies.**
- **Dissection.**
- **Emboli.** [from Lt side of the heart] → healthy Collaterals → healthy artery
- **SPASM OF THE CORONARY** (good prognosis) نقص ويرجع باك

مأش في حصة
sublingual nitrates

2. NON – CORONARY ARTERY DISEASE: (LOW CO)

- Any cause of low CO, especially: AS. to symp low CO sympt.

b) Decreased quality of coronary blood: (Hypoxic Blood)

- Anemia.
- Cyanotic conditions (hypoxia).

2) Increased myocardial oxygen demand:

- ↑ myocardial contractility: e.g. Ventricular hypertrophy.
- ↑ preload: e.g. Hyperdynamic circulation.
- ↑ afterload: e.g. Systemic hypertension.
- ↑ HR: Tachycardia.
- SEVERE STRESS: (physical, mental).

من أشهر الأدوية
التي تقلل الـ work
= β-blockers.

ATHEROSCLEROSIS

a disease of Big Pulsating Vessels

1. What is atherosclerosis ??



It is a process in which **lipids**, particularly **cholesterol**, are **deposited** in: the innermost layer of the artery (the **intima**) causing **ATHEROMA**.

The atheroma causes **gradual narrowing** of the arterial lumen leading to: **ISCHEMIA** and necrosis of the tissues.

The already present **COLLATERALS** open to bypass the narrow segment.

2. Lesions of atherosclerosis:

① Asymptomatic
Characteristic

Early Lesion

(The fatty streak) Contains 2 types of cells:

- o Foam cells : macrophages filled with lipids. (engulf lipids LDL)
- o T-lymphocytes: and some smooth muscle cells.

② Angina

Advanced lesion

(The fibrous plaque)

- o Thickened cap of dense CT.
- o Large number of smooth muscle cells.

③ AMI

Far advanced lesion (The complicated lesion):

- o Degeneration, calcification.
- o Ulcers, cracks, fissures.....and: **PLAQUE RUPTURE**.
- o **Occlusion** of the lumen due to: plaque rupture or thrombosis.

less common

More Common

3. Complications of atherosclerosis:

- Coronary Artery Disease (CAD). (Angina, AMI)
- Cerebro - vascular Disease (CVD). (TIA, stroke)
- Peripheral Arterial Disease (PAD).

The lesions of atherosclerosis

1. Early lesion

(The fatty streak)

triglycerides
cholesterol : LDL
Asymptomatic

Asymptomatic
طبيب في البيت



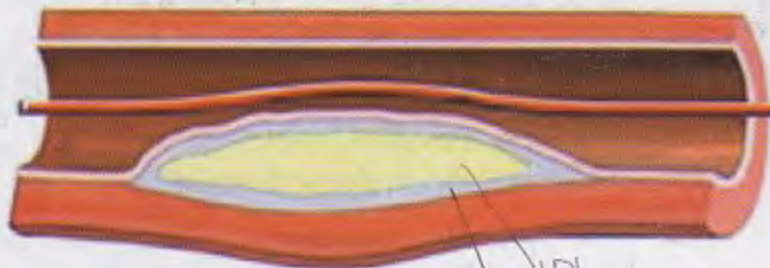
fat deposition
Foam Cells :
⊕ Growth Factors : ↑ proliferation of smooth Ms

2. Advanced lesion

(The fibrous plaque)

Angina
سبب

Plaque with
fibrous cap



Pass on
smooth
surface

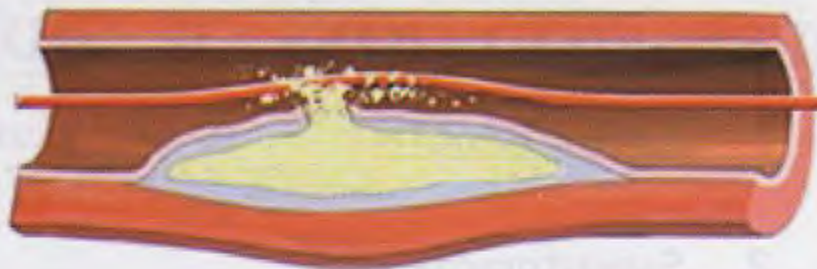
LDL
Fibrous plaque : CT & smooth Ms

3. Far advanced lesion (The complicated lesion)

AMI
تفوق

Cap
ruptures

ulcers, cracks, fissures
(By HTN)



Pass on
rough
surface



أي تعوية لانس الدم يتجلط

thrombosis on top of atherosclerosis

Blood clot forms
around the rupture,
blocking the artery



4. Risk factors for atherosclerosis:

1. Non – modifiable:

GAS

- Age: usually above 40 years.
- Sex: male : female = 5 : 1. (estrogen) ↑HDL
- Type A personality: nervous, intellectual.
- Genetic factors: positive family history.

2. Modifiable:

a) High risk:

DM
D

HTN

- DYSLIPIDEMIA: ↑ total cholesterol, ↑ LDL, ↓ HDL. (LDL ↑, HDL ↓)
- Diabetes mellitus refer to DM.
- Hypertension: causes endothelial damage
- Cigarette smoking. (endothelial damage)

b) Less risk:

بؤليله

Pu

H براكه

كلكه

- Obesity. ↑ DM, Hypertension, dyslipidemia, inactive diet rich in SFA
- Diet: rich in saturated fat & cholesterol.
- Physical inactivity.
- Psychological stress.
- Hyperuricemia. → uric acid is toxic to endothelium
- Homocysteinemia. ↑ incidence of coagulation (thrombus)
- Heavy alcohol intake. →
- CCPs. → hypercoagulability

PRESENTATIONS OF IHD

1. Asymptomatic: (silent myocardial ischemia)

- More common in DIABETES. (due to diabetic neuropathy)

2. Symptomatic:

a) Typical presentation: (chest pain)

- Angina pectoris or acute myocardial infarction.

b) Atypical presentation:

MAC + PS₂

S.P.C.A.M

- Congestive heart failure. Rt on top of Lt sided heart failure
- Cerebrovascular stroke. Necrotic stroke → mural thrombus → embolus
- Arrhythmias. (ischemia)
- Acute indigestion. severe dyspepsia, inferior infarction
- Syncope. low CO
- Sudden death. ICA occlusion serious arrhythmia
- Peripheral embolism.
- Marked fatigue. low CO

Homocystinemia ↑ Homocystien

- ↳ Hereditary (inborn error of Metabolism)
- ↳ Acquired

↳ ↓ Vit B₆ (Pyridoxin)

↳ ↓ Vit B₁₂ ()

↳ ↓ Vit B₉ (folic acid)

Homo ——— B₆
 B₁₂ ———> metabolite
 B₉

↓
Homocystinemia

What causes the pain??

CLINICAL PICTURE

SYMPTOMS:

"CHEST PAIN"

Site & character

diffuse retrosternal, radiating

Sharp, severe, tearing, especially at night

Not relieved by

analgesics, but relieved by

Character & duration

Continuous & severe

Severe cases III > 20 min, AMI or IMA should be considered

Associated signs

Swelling, tenderness and redness

of chest wall, especially at night

Associated signs

Swelling, tenderness and redness

of chest wall, especially at night



Etiology: IHD

angina

Cardiologic Chest Pain

Caused by Myocardial ischemia
lasting for few minutes

Not resulting in Myocardial necrosis

PRESENTATIONS OF IHD

1. Asymptomatic (silent myocardial ischemia)

More common in Diabetics

2. Symptomatic

a) Typical angina

Anginal pattern of severe myocardial ischemia

b) Atypical angina

Cardiac chest pain

Cardiac chest pain

Angina

Atypical

Angina

Angina

Angina

Angina

ANGINA PECTORIS

دورة غير نه
[Stage II atherosclerosis]

→ Pectoral region

DEFINITION

- Attacks of chest pain, caused by myocardial ischemia, usually lasting for several minutes, and: **not resulting in myocardial necrosis.**

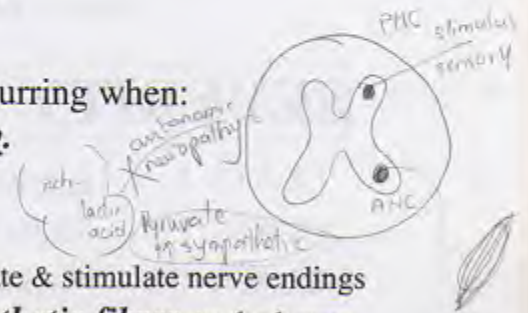
ETIOLOGY

No Pain sensors in Myocardium

- Angina occurs due to brief myocardial ischemia occurring when:
The myocardial oxygen demand exceeds the supply.

- What causes the pain ??

- When myocardial hypoxia occurs, metabolites accumulate & stimulate nerve endings in the myocardium to discharge impulses along **sympathetic fibres** to the lower cervical & upper thoracic segments of the spinal cord with subsequent radiation to the corresponding dermatomes, producing sensation of pain coming from these areas.
- Pain may be ABSENT in DIABETES (Silent ischemia):
This is due to autonomic neuropathy.



mainly
H₂O₂

CLINICAL PICTURE

1 SYMPTOMS: "CHEST PAIN" "Typical"

- Site & reference: diffuse retrosternal, radiating to:
 - Shoulders, arms & forearms (especially the left).
 - Neck and lower jaw.
 - Less commonly: back or epigastrium. (inferior infarction)
- Character & duration:
 - Crushing, Compressing, Squeezing, Suffocating,
 - Several minutes (if > 20 min, AMI or UA should be considered).
- Precipitated by: ↑ demand
 - **S**tress (physical and emotional), **S**exual intercourse.
 - Cold weather, ↑ BMR + VC → ↑ afterload Heavy meals.
- Relieved by: rest, SL nitrates.
- Associated symptoms:
 - Dyspnea, palpitation, dizziness.
 - Sweating, nausea, vomiting.
 - ANGOR ANIMI: Sense of impending doom.

التشخيص بواسطة
كل من العيان + القسطرة

Non-specific

maybe due to
Cardiac muscle

احساس بالوفاة

2 SIGNS: "There may be no abnormality"

- General signs: (during the attack)

- Pallor, sweating. (General manifestations)
- Transient tachycardia and hypertension. (Pulse + Bl. Pr.)

- Cardiac signs:

- S₁: Weak. (Segmental Muscular weakness, not all muscular component lost)
- S₂: Reversed splitting. (Turbulent flow)
- S₄: (due to decreased ventricular compliance by ischemia).
- Signs of complications: e.g. systolic murmur of MR (pap. mus. dysfunction).

SEVERITY

"4 classes of angina according to severity"

CLASS I: Angina on marked activity, nothing occurs on ordinary activity.

CLASS II: Slight limitation of physical activity on ordinary effort.

CLASS III: Marked limitation of physical activity on less than ordinary effort.

CLASS IV: Angina may be present even at rest.

TYPES *Clinical*

1. Stable or Classical angina:

(typical form)

Exertional = Effort

- The pain is relatively constant as regards severity, precipitating factors and relief.

2. Unstable angina: 20%

- ① Worsening angina (crescendo angina): increased frequency, severity or duration of a preexisting stable angina.

- ② Angina starting to occur at REST (or with minimal exertion).

- ③ Angina of RECENT onset (within 2 months).

- ④ Angina RESISTENT to therapy.

- ① It carries a bad prognosis as 20% will pass to AMI within several months.

- ② It is considered an intermediate syndrome between stable angina and AMI.

3. Variant angina:

(Prinzmetal, Vasospastic angina)

- ① Angina at rest: caused by SPASM of a coronary artery (normal or atherosclerotic), and is not precipitated by increased oxygen demand.

- ② ECG:

Transient ST elevation.

- ③ Ambulatory ECG:

Diagnostic.

- ④ Positive ergonovine provocative test:

Diagnostic. challenge test

- ⑤ Coronary angiography:

[atherosclerosis] Normal or narrowed lumen.

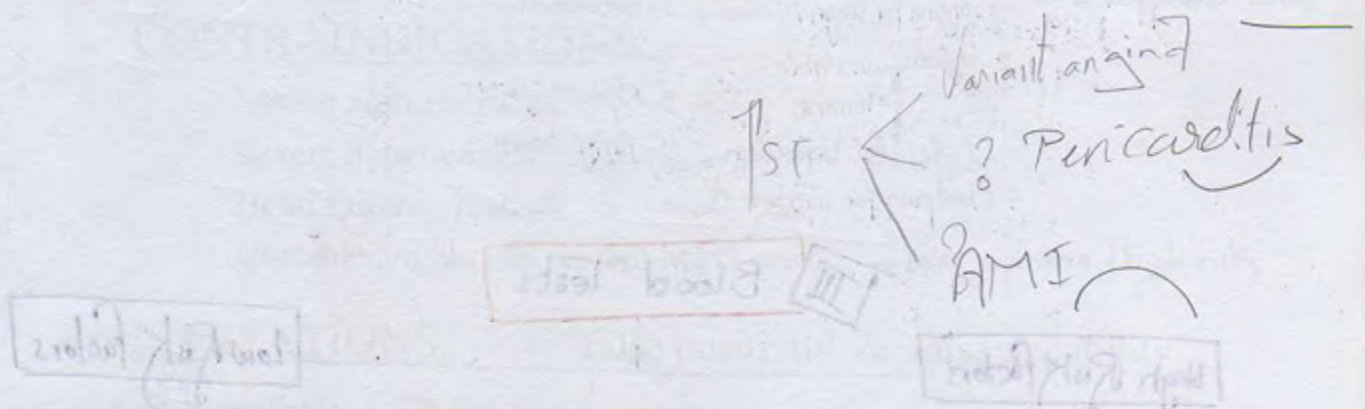
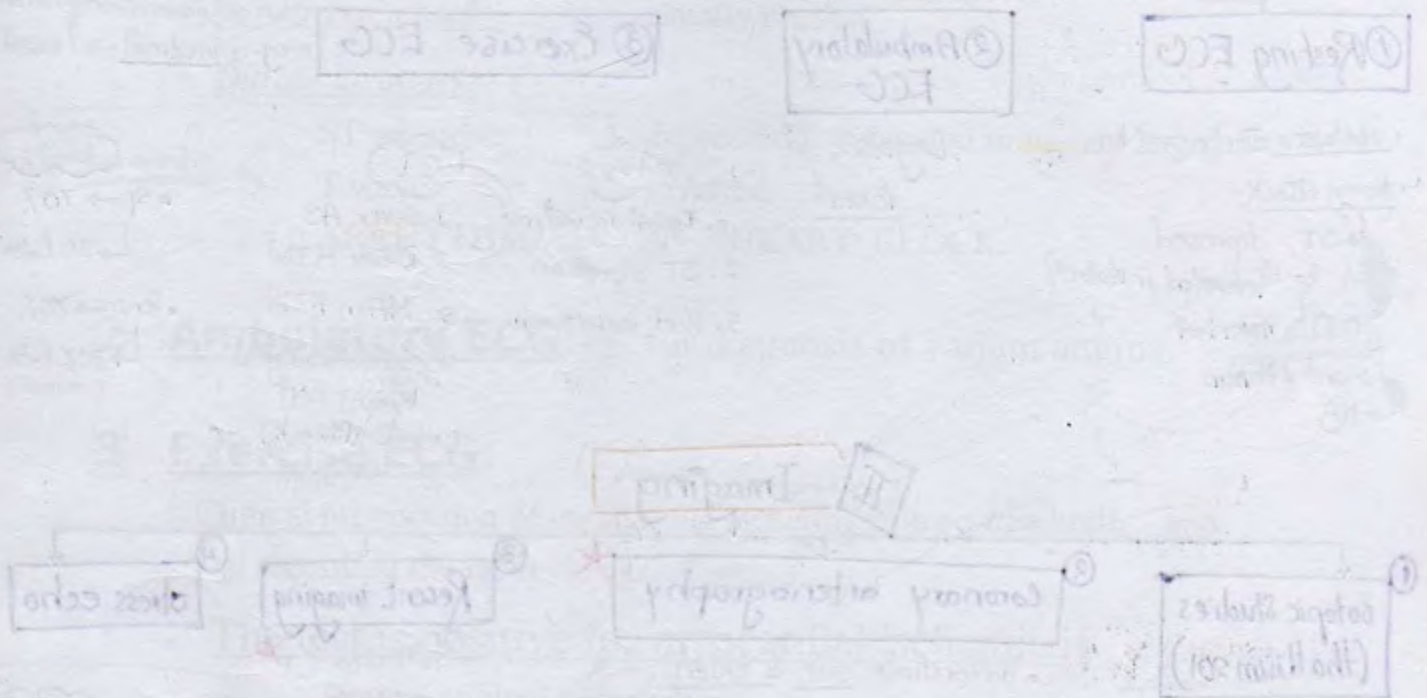
4. Other forms of angina:

(uncommon)

- Nocturnal: it awakens the patient from sleep (due to ↑ sympathetic activity related to dreams).
- Angina decubitus: occurs on lying down and relieved by sitting (occurs in heart failure).
- Angina of Lewis: in cases of aortic regurge, it is nocturnal.

NB

Variant Angina
 Catheterization
 Vascularitis (arteritis)
 القسطنطينية من هجره للقصيرين
 بقصور شرايين التوي
 مش فيه اهد



Investigations Angina

I ECG

① Resting ECG

- inbetween attacks = N.
- during attack:
 - ST: depressed (elevated in Variant)
 - T: inverted
 - arrhythmia
 - HB

② Ambulatory ECG

Diagnostic in Variant

③ Exercise ECG

Clinical provocation of ischemia using a treadmill & record ECG

+ve

1. Typical anginal pain
2. ST depression
3. Vent. arrhythmia

C.I.

1. Severe AS
2. Severe HTN
3. HF or HTN
4. Unstable angina Recent AMI (1 week)

Limits

- sp → 70%
 - ↳ 30% false +ve
- Sens. → 70%
 - ↳ 30% false -ve (missed)

II Imaging

① Isotopic Studies (thallium 201)

- uptake by healthy
- Cold spot appear at Exercise & disappear at rest

② Coronary arteriography

- demonstrate site & extent

degree of narrowing

indications

1. angina for surgery [Coronary revascular.]
2. angina:
 - unstable
 - Variant
 - Post. infarction
3. Chest pain for uncertain et.

Complications

1. Embolization
2. dissection
3. Rupture → MI
4. Arrhythmia

③ Recent imaging

- Multi slice CT
- MRCA
- IVUS

④ Stress echo

methods

- exercise
- IV dobutamine

Result

RWMA "hypokinesia"

III Blood Tests

High Risk Factors

- Dyslipidemia
- DM

Low Risk Factors

- Homocysteinemia
- Hyperuricemia

INVESTIGATIONS

ECG : angina متشكك في ال ECG
even in the attack

1 Resting ECG:

• In between attacks: usually normal.

• During an attack:

non-specific ← ST segment: depressed (elevated in variant angina).
T wave: inverted.
- ARRHYTHMIAS or HEART BLOCK.

2 Ambulatory ECG: * for diagnosis of variant angina.

ممتاز في التشخيص
"Excellent"

3 Exercise ECG:

(اختبار تنشيط في راحة)

"Clinical provocation of myocardial ischemia using a treadmill, and
and recording the ECG changes"

• The test is positive for myocardial ischemia if: أي واحدة من الثلاثة

- Typical anginal pain occurs.
- ST segment depression occurs.
- Ventricular arrhythmias occur.

• CONTRAINDICATIONS:

- Severe aortic stenosis. See AS.
- Severe hypertension. (not regular physical activity)
- Heart failure. (Bed rest + أمثلة: ضعف)
- Unstable angina & recent MI (1 week). → severe ischemia is high risk

High power of contraction
to overcome Afterload

• LIMITATIONS: "false positivity & false negativity"

a) Specificity: (70 %)

- % of patients without CAD who have negative test (true negative).
- Therefore there are 30 % false positive. (30% with other disease's not CAD but true)

b) Sensitivity: (70 %)

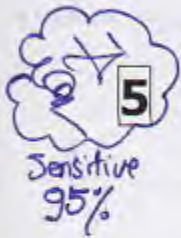
- % of patients with CAD who have positive test (true positive).
- Therefore there are 30 % false negative. (30% missed) detected by the Ex. ECG

Hence a negative test does not adequately rule out CAD

* Holter monitor: named after its inventor, Norman J. Holter (1914-1983), American.

4 Isotopic studies: (Radioactive Thallium - 201)

- Thallium (201) is taken up by healthy myocardium & not by the ischemic myocardium.
- Ischemia appears as a filling defect (COLD SPOT) at exercise & disappears after rest.



5 Coronary arteriography:

- It demonstrates the site and extent of coronary occlusion:

Indications:

- Angina which is: indicated for coronary revascularization.
- Angina which is: unstable ^{high risk} variant & post-infarction angina.
- Recurrent chest pain of uncertain etiology.

Complications:

- Myocardial infarction. (Rupture of the Coronary : Mechanical)
- Arrhythmias. (any Cardiac Manipulation)
- Embolization. (detachment of atheroma eg in aorta)
- Arterial dissection. (Mechanical injury to intima)

6 Recent imaging: Not widely used : V. expensive

V. thin slice

- Multi-slice CT, Magnetic resonance coronary angiography (MR - CA).
- IVUS intra vascular ultrasound what is IVUS ???
↳ Can describe plaque rupture vulnerability

7 Stress echocardiography:

V. minor Role

• Methods:

- Exercise. (to induce ischemia)
- IV dobutamine: for people who are unable to exercise. "Sympathomimetic"

• Result: "as it's segmental not global disease"

the same cold spot in thallium 201

- May show RWMA in ischemic areas (Hypokinesia).
Regional wall Motion abnormality

8 Biochemical screening:

(Blood tests) Risk factors

- For identification of risk factors such as:

- Dyslipidemia: lipids (TC, LDL, HDL, TG).
- Hyperglycemia: glucose. (DM)
- Hyperuricemia: uric acid.
- Homocysteinemia: homocysteine.

High risk

Low risk

L.O.

DIFFERENTIAL DIAGNOSIS

20
mark

لو سوال

قلب طمه عن كل عنوان
"most imp. criteria"

وبعد كده Scheme ايزاي تشخيص

- Causes of: acute chest pain

- Myocardium: Angina, AMI.
- Pericardium: Acute pericarditis.
- Endocardium: Mitral valve prolapse.
- Aorta: Dissecting aortic aneurysm.
- Pulmonary: Acute pulmonary embolism.
- Pain of cardiac neurosis.
- Acute pleurisy.
- Acute massive lung collapse & pneumothorax.
- Oesophageal pain: GORD.
- Peptic ulcer & cholecystitis.
- Herpetic pain (Herpes zoster).
- Musculo-skeletal disorders e.g. Tietze syndrome.

TREATMENT

1 DURING THE ATTACK (Emergency)

1/ Complete REST.

2/ SUBLINGUAL DRUGS:

<u>Nitrates:</u>	Nitroglycerin	0.5 mg	} <u>Sublingually</u>
	Isosorbide dinitrate	5 mg	

These drugs act by:

- a) Dilating the coronaries.
- b) Reducing the work of the heart through: venodilator effect.
↓ pre load (more Venous)

Coronary Revascularization

Treatment of Angina

I in between attacks

General

- advised**
- Moderate life style
 - Smoking
 - Diet: ↓ SFA, chol.
 - Regular Exercise:
 - ↑ collateral coronary flow
 - ↑ HDL "improve lipid profile"
 - ↓ BP, ↓ JNR, ↓ O₂ demand
 - ↓ coagulability, ↓ fibrinolysis
 - ↓ platelet aggregation
- Causes**
- Risk Factors: HTN, DM, Dyslipid

During Attack

- Rest (complete)
- SL nitrates
 - ↓ dilate Coronaries
 - ↓ preload (venodilator) (work)

Pharmacologic

Anti Anginal Drugs



Nitrates

- dilate Coronaries
- ↓ preload (venodilator)

CCB

- dilate Coronaries (esp. in VA)
- ↓ after load
- ↓ contractility

β-Blockers

- ↓ BP, ↓ JNR
- ↓ contractility

Antiplatelets

to guard against AMI esp UA

- Aspirin 75-300mg/day
- Dipyridamol 75mg bid
- Ticlopidine 250mg bid
- Clopidogrel 75mg/day

Anticoagulants

Heparin IV (given esp aspirin to patients esp UA)

Action

Preparations

- Nitroglycerine 25mg/8hrs oral or TP
- Isosorbide dinitrate 20mg bid or TP
- Isosorbide mononitrate 20mg bid or SR 100mg once daily

S.E.

- Severe Headache
- Tolerance:
 - smallest eff. dose
 - allow free nitrate interval

Headache

- Flushing
- ↓ BP, ↓ JNR (esp HT)
- LL edema (reversible)
- JNR: nifedipine
- JNR: diltiazem

Brachospasm

- ↓ BP, ↓ JNR (fatigue M. HE)
- Mask signs of hypoglycemia in DM
- ↑ JNR: nifedipine
- ↑ JNR: diltiazem

Triple Therapy

- Nitrate
 - Nitrate + βB
 - Nitrate + CCB
- (NB) VA → CCB + Nitrates

At Dipyridamol

- advised: Diet (SFA, chol.) & regular Exercise
- Drugs

Drug	Action	Side Effect
Statins	↓ cholest. (liver synth.) ↓ LDL	↑ liver enz. Myopathy
Ezetimibe	↓ cholest. (intest. absorp.) ↓ LDL	↑ liver enz. Myopathy

Simvastatin + Ezetimibe = INEG

Cholestyramine	↓ cholest. (bind and sequester) ↓ LDL	N.V. Constipation
Fibrates	↓ TG	↑ myopathy & statin
Nicotinic acid	↓ TG ↓ LDL ↑ HDL	Dizziness headache Flushing

2 INBETWEEN THE ATTACKS

I. GENERAL MEASURES

a) TTT of the cause & correction of the risk factors:

- Cause: e.g. Vasculitis, AS, HTN.
- Risk factors: e.g. DYSLIPIDEMIA, * Diabetes, HTN.

تسريع
acceleration
of atherosclerosis

عوامل
Predisposing
Factors

b) Non – pharmacologic therapy:

نصائح

- Moderation of life: Avoid as possible the precipitating factors.
- Smoking: must be stopped.
- Diet control: Restriction of cholesterol & saturated fat.

• Physical activity: “REGULAR EXERCISE”

تدريبات

Benefits:

- ① Improves the collateral coronary blood flow.
- ② Improves the lipid profile. (↑HDL) [صحيح دواء يرفع HDL]
- ③ ↓ the heart rate, ↓BP & ↓myocardial O₂ demands.
- ④ ↓ platelet aggregation, ↑ fibrinolytic activity. (↑fibrinolysis)

II. SPECIFIC MEASURES

“Pharmacologic therapy”

دواء

أهداف العلاج

AIM:

- To ↑ myocardial O₂ supply (dilate the coronary arteries) → Nitrates
- To ↓ myocardial O₂ consumption (↓ work of heart), → βB → CCB

BY USING ANTI – ANGINAL DRUGS.

- To guard against the development of AMI (thrombosis),

BY USING ANTIPLATELETS and / or ANTICOAGULANTS.

* TTT of Dyslipidemia is mentioned later.

DRUGS:

1) ANTI-ANGINAL DRUGS: "Nitrates, β B, CCBs"

a) Nitrates:

* Actions:

- Dilate the coronary arteries.
- Reduce preload. [$\downarrow$ work of heart]

* Preparations:

- Nitroglycerine: 2.5 mg / 8 hours orally, or TP. ^{topical patches}
- Isosorbide dinitrate: 20 - 40 mg twice daily, or TP. ^{once daily}
- Isosorbide mononitrate: 20 mg twice daily, or SR 100 mg, OD. ^{sustained release}

* Side effects:

- Severe headache. (severe vaso dilatation)
- Tolerance (hyporesponsiveness): = tachyphylaxis

o Use the smallest effective dose.

o Allow a nitrate-free interval ($\approx$ 8 hs)

1 tab 8 a.m.

1 tab 4 p.m.

X no in 12 a.m.

angina v. rare in sleep to occur

لو حصل ياخرباه تحت اللسان

b) β - blockers: (β B)

* Actions:

- $\downarrow$ HR & $\downarrow$ BP.
- $\downarrow$ myocardial contractility.

$\downarrow$ Cardiac work

* Preparations:

- Non-selective: Propranolol, 60 mg / 8 hours. ^{non-selective}
- Cardio-selective: Atenolol, 50 - 100 mg / day. ^{Cardio selective}

* Side effects:

- Chest: Bronchospasm. Both: Not selective 100% ^[أكثر حاجة بخاف منها]
- Heart: Bradycardia, Hypotension, Heart block.
- Others: Fatigue, Reversible impotence, Masking signs of hypoglycemia in diabetes.

c) Calcium channel blockers: (CCBs)

* Actions:

- Dilate the coronary arteries (extremely effective in V ^{variant} Angina)
- Reduce afterload.
- Reduce the myocardial contractility.

لو قلب HF $\leftarrow$ Stop

* **Preparations:**

- Verapamil 80 mg / 8 hours.
- Diltiazem 60 mg / 8 hours.
- Nifedipine 10 mg / 8 hours.
- Amlodipine: 5 mg / day.

* **Side effects:**

- Headache & flushing. (arterial vasodilatation)
- Hypotension & aggravation of heart failure. (ppt. factor of HF)
- Bradycardia: with verapamil & diltiazem. (AV Block!)
- Tachycardia: with nifedipine. (Reflex tachy due to severe hypotension)

V. common

← REVERSIBLE OEDEMA OF LLs. [↑ Capillary permeability]

Choice of drug therapy

1. Begin with nitrates, if there is poor response..... THEN: 1
2. For stable exertional angina: add β B or CCB to nitrates. 2
3. For recurrent angina on 2 drugs: apply triple therapy. 3
4. For variant angina: add CCB to nitrates.
5. For unstable angina: **heparin & aspirin** are most important.

2) ANTIPLATELETS & ANTICOAGULANTS:

- **Aspirin** (low dose): 75 – 300 mg / day (single dose).
- **Dipyridamole:** 75 mg twice daily.
- **Ticlopidine:** 250 mg twice daily.
- **Clopidogrel:** 75 mg once daily.

clavix

They protect against AMI & death especially in patients with UA

(1^{ry} prophylaxis)?

• **Heparin:**

- IV heparin (with antiplatelets) is given in UA.

{aspirine}

الأسبرين
4000 E/month

oral/written

TTT OF DYSLIPIDEMIA

A) LIFE – STYLE MODIFICATION

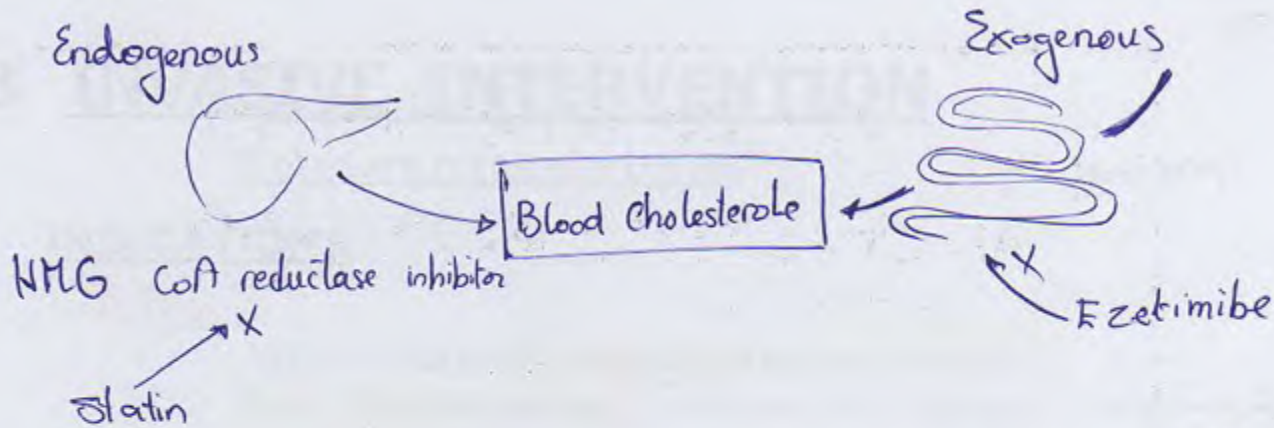
- Diet control.
- Regular exercise. $\uparrow$ HDL

B) DRUGS ¹

DRUG	ACTION	SIDE EFFECTS
STATINS	$\downarrow$ cholesterol ($\downarrow$ hepatic synthesis) $\downarrow$ LDL	Liver: $\uparrow$ enzymes Muscle: Myopathy (Rhabdomyolysis)
EZETIMIBE	$\downarrow$ cholesterol ($\downarrow$ intestinal absorption) $\downarrow$ LDL	Liver: $\uparrow$ enzymes Muscle: Myopathy
SimvaSTATIN + EZETIMIBE = INEGY		
Cholestyramine ملوش قير	$\downarrow$ cholesterol (Bile acid sequestrant) $\downarrow$ LDL Binds bile acid $\rightarrow$ stool Blood cholesterol try to compensate $\rightarrow$ $\downarrow$ cholesterol	N, V, constipation also NB: used in jaundice
FIBRATES	$\downarrow$ TG	$\uparrow$ risk of severe myopathy with Statins
Nicotinic acid الو حيد لك احسنه	$\downarrow$ LDL, $\downarrow$ TG $\uparrow$ HDL	Headache Diarrhea Flushing

Exercise

¹ How would you deal with combined dyslipidemia ??



Rhabdomyolysis = acute Massive sk. Ms destruction (Renal Failure)

★ tt of Combined dyslipidemia: $\begin{cases} \uparrow \text{Cholesterol} \\ \uparrow \text{TG} \end{cases}$

(NB) Can't give Statin + Fibrates
 ↳ Rhabdomyolysis may occur.

ممكن الـ statin مع fibrates

→ Hypertriglyceridemia >400 too dangerous ACUTE complication
 ↳ pancreatitis (fatal)
 ↳ Retinal V. thrombosis

Start with Fibrates then shift to Statins

if Hyper TG >200 bas: Start with Statins

(NB) uses of β -Blockers

① HF

② MVP

③ Angina

④ HTN

(NB) A-V Block

β -blocker

CCB (Verapamil, Diltiazem)

Digoxin

3 INVASIVE INTERVENTION

"Coronary revascularization"

(إعادة توصيل الدم)

INDICATIONS

1. General:

- Angina which is not responding to medical treatment.
- Post – infarction angina: *to improve the prognosis.*

مش هتفنى لما قلبه
كله يموت

2. Specific to the technique: *see below.*

TECHNIQUES

1. PCI

(**P**ercutaneous **C**oronary **I**ntervention)

[PTCA + Stent (**P**ercutaneous **T**ransluminal **C**oronary **A**ngioplasty)]

- Introduction of a balloon to dilate the stenotic artery.
- Introduction of an intra – luminal **Stent** to maintain patency of the dilated artery.

o Indications:

- Stenosis of one or 2 vessels only (except left main coronary artery).
- Stenosis of bypass grafts following CABG.

o Complications:

- Complications of coronary arteriography (mentioned before).
- Acute coronary occlusion. (*detached emboli distal*) [so we put filter]
- Restenosis.

2. CABG

Surgical

(**C**oronary **A**rtery **B**ypass **G**raft)

- Grafting a segment of saphenous vein or an **IMA** between:
the ascending aorta & the coronary artery distal to the obstruction.

o Indications:

- Stenosis of 3 or more vessels.
- Stenosis of the left main coronary artery.

o Results:

- In 90 % of patients: *Excellent relief of angina.*
- In 10 % of patients: *Restenosis of the vein graft in the first year.*

3. Others:

(Laser angioplasty).



ACUTE MYOCARDIAL INFARCTION

DEFINITION

Ischemic necrosis of part of the myocardium due to:

"Sudden & persistent cessation of its blood supply" (early stage III atherosclerosis)

ETIOLOGY

- Refer to IHD.

SITE

"Depends on the occluded artery" See P143

1. Occlusion of the left anterior descending artery:
 - Causes anterior infarction.
2. Occlusion of the circumflex artery:
 - Causes lateral infarction.
3. Occlusion of the right coronary artery:
 - Causes inferior infarction.

CLINICAL PICTURE

Symptoms [Chest Pain]

① Patients presents in early morning
② Bronchial asthma
③ AMI

Onset:

Usually in the early morning due to:

- ① Rise in plasma catecholamines. ↑ sympathetic ↑ work
- ② Rise in plasma cortisol. → H₂O retention ↑ blood (Circadian rhythm)
- ③ Increased platelet aggregation. (Catecholamine induced)



Pain:

(the main presenting symptom)

a) Typical: as anginal pain, BUT:

[ألم في الصدر أو الذراع]

- ① More severe, more prolonged, more radiating.
- ② May occur without precipitating factors.
- ③ Not relieved by rest or sublingual nitrates.

Morphine ٢-٤ ملغ

b) Atypical:

- Sense of indigestion. (inferior infarction)
- Atypical location of pain. eg: inside of radiation without original site

④

c) Absent: (silent myocardial infarction)

Autonomic neuropathy, e.g. DIABETES.



- ELDERLY PATIENTS. (degeneration)
- During anesthesia or in a transplanted heart. (denervated)
- Disturbed consciousness level, heavy sedation, shock.

- DCL

tissue hypoperfusion
→ ischemia in nerve
(in bl. in vasa nervosa)

White Knight Love

Signs

1. General:

a) General appearance:

- Pale, sweaty, restless.

b) Pulse:

• RATE:

- Tachycardia
- Bradycardia

(sympathetic hyperactivity).

(HB or ↑ vagal tone due to pain).

• RHYTHM:

Arrhythmias. (ischemia)

• PULSE VOLUME:

may be small due to LVF or shock.

(Pain & Cardiacogenic)

c) Blood Pressure:

- Initial hypertension (sympathetic hyperactivity).

- Hypotension (LVF or shock or ↑ vagal tone due to pain).

⊙ Decapitated BP:

marked drop of SBP with slight drop of DBP.

↓ CO

↓ Pulse Pr.

Some resistance

d) Fever:

(non specific response to tissue necrosis) [Pyrogens]

- Moderate rise within 1 – 2 days of the onset and may last for 1 week.

2. Cardiac:

- S₁: Weak.

- S₂: Reversed splitting.

- ⊕ S₃: due to flabby myocardium. if = tachy = Gallop

- S₄: due to decreased ventricular compliance by infarction.

- ⊕ Signs of complications: e.g. systolic murmur of MR (pap. mus. rupture).

no dysfunction

- ⊕ Pericarditis

COMPLICATIONS

1 Early Catastrophic Complications: (first few days)

1. Sudden death:

due to,

- VF.
- Rupture of the weak myocardium.
- Occlusion of the left main coronary artery.

2. Cardiac arrhythmias:

all types may occur,

- The most common are: extrasystoles. (rather benign)
- The most serious are: VT & HB.

3. Cardiac failure:

- Acute LVF. = acute pulmonary edema

4. Shock: (و افتاد و احاطت بـ AMI قلوب shocked و لم ينجحوا في علاجها) (HR) (HR) (HR)

a) Cardiogenic: 100% mortality

- Caused by massive infarction, leading to pump failure.
- There is hypotension & tachycardia. (Reflex: Mary's)
- Prognosis: very bad.

b) Neurogenic:

- Caused by severe pain, leading to vagal stimulation.
- There is hypotension & bradycardia.
- Prognosis: good. "morphine" 2-4 mg

5. Myocardial rupture: necrotic weak part

- Rupture of ventricular free wall → cardiac tamponade & death.
- Rupture of the septum → acquired VSD.
- Rupture of the papillary muscles → acute MR.

6. Pericarditis: → early & late: pleuropericarditis

- Usually dry, but effusion may develop. (pericardial Rupture)

Pain in shoulder $\rightarrow$ Sympathetic (long weak pain)

$\rightarrow$ arterial spasm

$\rightarrow$ ischemia of CT in shoulder joint

$\rightarrow$ Local release of growth factors

$\rightarrow$ fibroblasts $\rightarrow$ fibrosis

$\uparrow$ must give
Corticosteroids

2 Late Complications

(after several weeks)

1. Post infarction angina & recurrent infarction:

- Due to affection of other coronaries. 99% arteriosclerosis

لضعف الشرايين
معدود
do angiograph
do surgery → PCI
CABG

2. Post infarction syndrome (Dressler's syndrome):

"Pleuropericarditis"

- Onset: developing weeks after infarction.
- Caused by: autoimmune response to damaged cardiac tissue.
- Responds to: corticosteroids.

pericardium
Ab → p laurus

3. Frozen shoulder (shoulder hand) syndrome:

"Pain & stiffness of left arm & shoulder"

- Onset: developing weeks after infarction.
- Caused by: reflex arteriolar spasm leading to ischemia & fibrosis.
- Responds to: corticosteroids & physiotherapy.

⊕ Csh factors
→ ⊕ fibroblasts

4. Thrombo – embolic complications:

- Mural thrombosis (in the LV) → systemic embolism.
- DVT (from prolonged recumbency) → pulmonary embolism.

in brain
→ CVS

(Bed rest)

5. Myocardial aneurysm:

- Clinical presentations:

- Recurrent embolisation. (stagnant bl.)
- Recurrent arrhythmias. (stretch)
- Refractory HF. (Mechanical Factor)
- Rupture of the aneurysm. → tamponade or sudden death

- On examination:

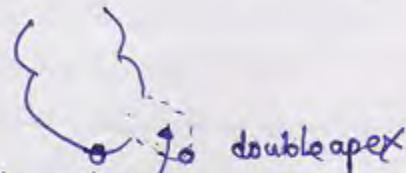
- Double apex.

- Investigations:

- ECG: persistent ST segment elevation > 6 weeks.
- Echo: diagnostic.

Follow up

Echo
ECG
Persistent ST elevation
for 6 weeks
Follow up



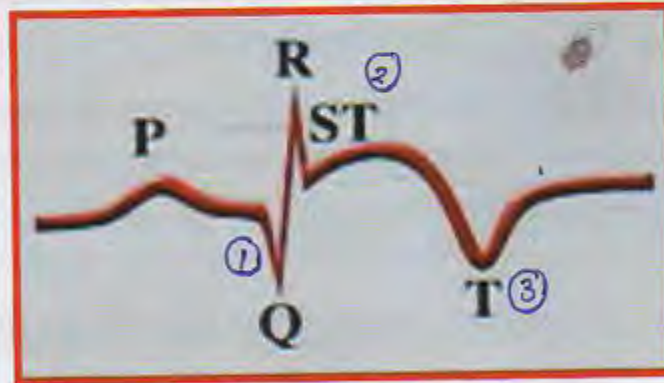
الأيض

INVESTIGATIONS

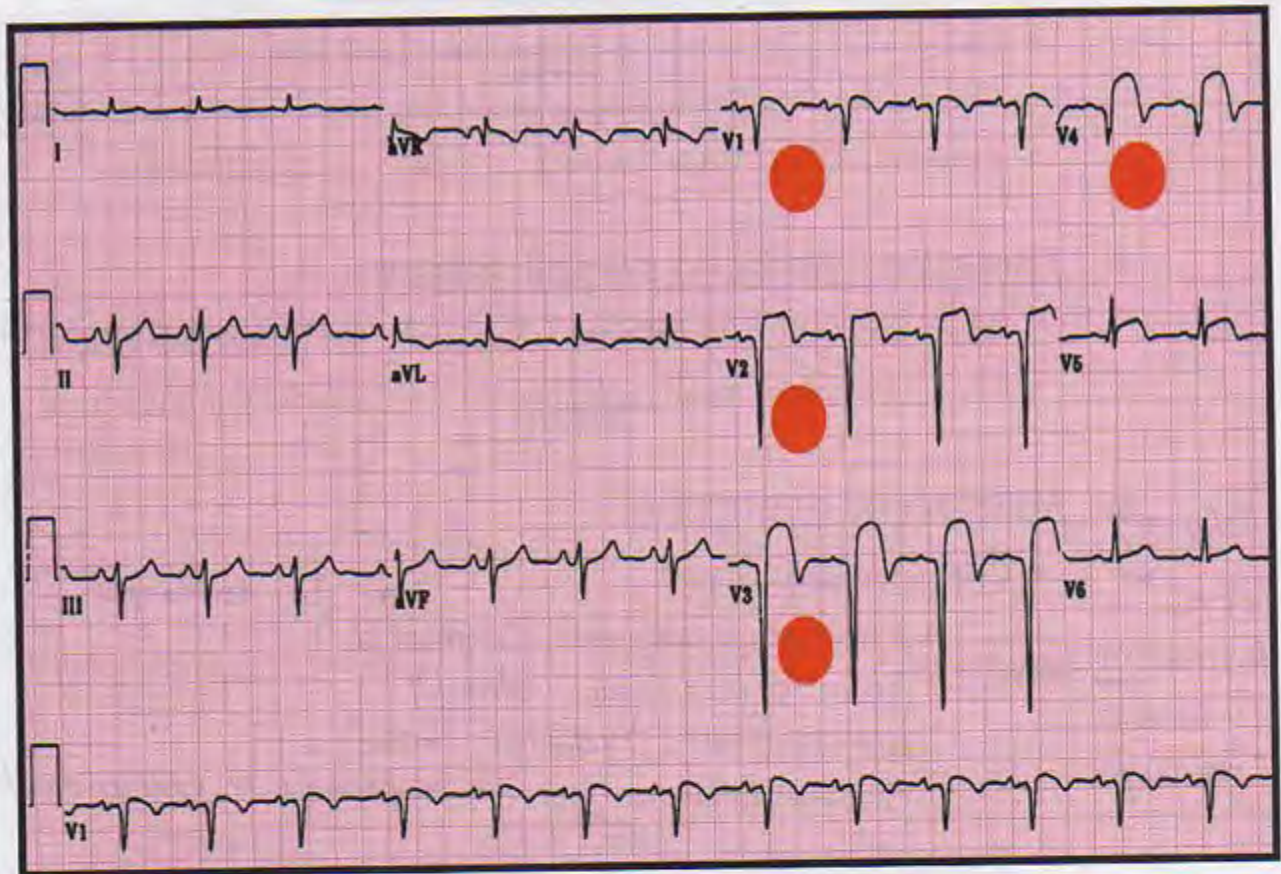
Hyperacute "T": tall & peaked
 → AMI: too early to be detected
 ↳ Hyperkalemia
 إذا لم يتغير يبق عيان مخطوط ويستعمل streptokinase

1 ECG:

- Inverted T wave = ischemia.
- Pathological Q wave = infarction.
- Elevated convex ST segment (injury pattern) = recent infarction.



D.D elevated ST
 AMI → Convex
 Variant angina → Flat
 Pericarditis → Concave



Myocardial infarction (Typical ECG pattern)

- Recent → (elevated ST)
- Anteroseptal → (V1 - V4) (LAD)

A normal ECG does not rule out acute myocardial infarction

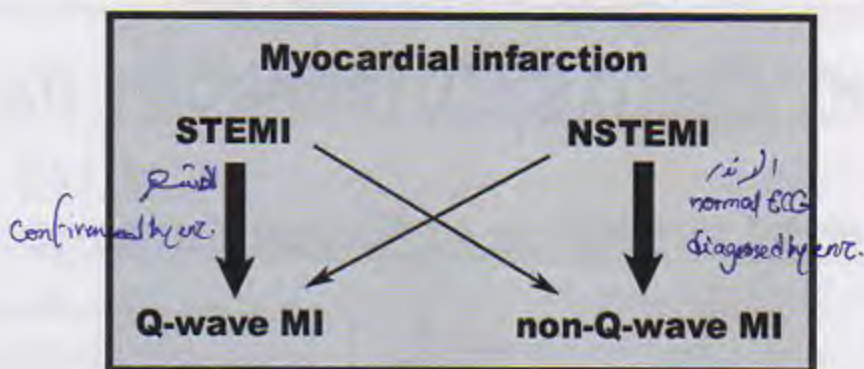
↳ diagnosed by Enzymes (NSTEMI, non Q-wave MI)

According to ST segment, there are 2 types of MI:

1. **STEMI:** **ST** segment **E**levation **M**yocardial **I**nfarction
[confirmed by cardiac markers (enzymes)]
2. **NSTEMI:** **Non ST** segment **E**levation **M**yocardial **I**nfarction
[diagnosed by cardiac markers (enzymes)]

According to Q – wave, there are 2 types of MI:

1. **Q – wave MI:** “Previously called: **Transmural MI**” whole thickness of the heart
[usually associated with: STEMI]
2. **Non Q – wave MI:** “Previously called: **Subendocardial MI**” ends & Myo not q1.
[usually associated with: NSTEMI]



According to affected leads, there are many sites of MI:

The constellation of leads with $\uparrow$ ST & pathological Q wave helps to identify what area of the heart is injured, & therefore helps predict the culprit artery:

Wall Affected	Leads Showing ST Segment Elevation	Suspected Culprit Artery
Septal	V ₁ , V ₂	LAD
Anterior	V ₃ , V ₄	LAD
Lateral	V ₅ , V ₆ , I, aVL	Circumflex
Anteroseptal	V ₁ , V ₂ , V ₃ , V ₄	LAD
Anterolateral	V ₃ , V ₄ , V ₅ , V ₆ , I, aVL	LAD or Circumflex
Extensive anterior	V ₁ , V ₂ , V ₃ , V ₄ , V ₅ , V ₆ , I, aVL	Left main coronary
Inferior	II, III, aVF	Right Coronary

Sudden death

2 CARDIAC MARKERS (Enzymes):

- Following AMI, the necrotic cardiac tissue will release several proteins:

a) Specific markers: "Typical enzymes"

- Troponins (I & T): ★ *E41* *More sensitive*
 - They start to rise within 4 – 8 hours & return to normal in 6 – 7 days.
- CK – MB: Creatine Kinase (Myocardial Band)
 - It is a specific subtype of the nonspecific CPK.
 - It starts to rise within 4 – 8 hours & return to normal in 2 – 3 days.

b) Non – specific markers: (old, not used)

- CPK. (also from sk. Ms)
- AST. (aspartate serum Transaminase; Liver)
- LDH. (lactate dehydrogenase; lung, sk. Ms)
- Myoglobin. (sk. Ms.)

3 General evidence of tissue damage:

- Leukocytosis
- ↑ ESR
- ↑ CRP (*hs-CRP*)

Acute phase reactants

4 Imaging:

a) Non-invasive:

1. Echocardiography:

- Akinesia of the infarcted area. (*at rest*) *RWMA*
- Complications, e.g. MR, Myocardial aneurysm, Mural thrombosis.

2. Isotopic studies:

- Thallium imaging: infarcted area appears as a cold spot.
- Technetium imaging: infarcted area appears as a hot spot.

b) Invasive: (Coronary arteriography)

- To demonstrate: the site of the occluded vessel.
- To assess: the condition of the other coronaries.

c) Recent imaging:

- Multi – slice CT, MR – CA.
- IVUS.

DIFFERENTIAL DIAGNOSIS

- From all causes of acute chest pain (refer to angina).

PROGNOSIS

الحالة المشغولة

BAD PROGNOSTIC CRITERIA IN AMI

1. PATIENT:

- Age: old.
- Heart: originally unhealthy weak or dilated cardiac muscle.
- Associated risk factors of atherosclerosis & CAD: e.g. DM, HTN.

2. INFARCTION:

- Site: extensive anterior is worse than lateral & inferior.
- Size: the larger, the worse the prognosis.

3. MANAGEMENT:

- Delayed: and inability to apply early thrombolytic therapy.
- Ineffective: Failure of the patient's condition to stabilize during the first few days of management.

4. COMPLICATIONS:

- Development of severe complications: e.g. cardiogenic shock.
- Persistent elevation of the ST segment in ECG. (Aneurysm)

NB ACUTE CORONARY SYNDROMES (ACS)

1. Unstable angina.

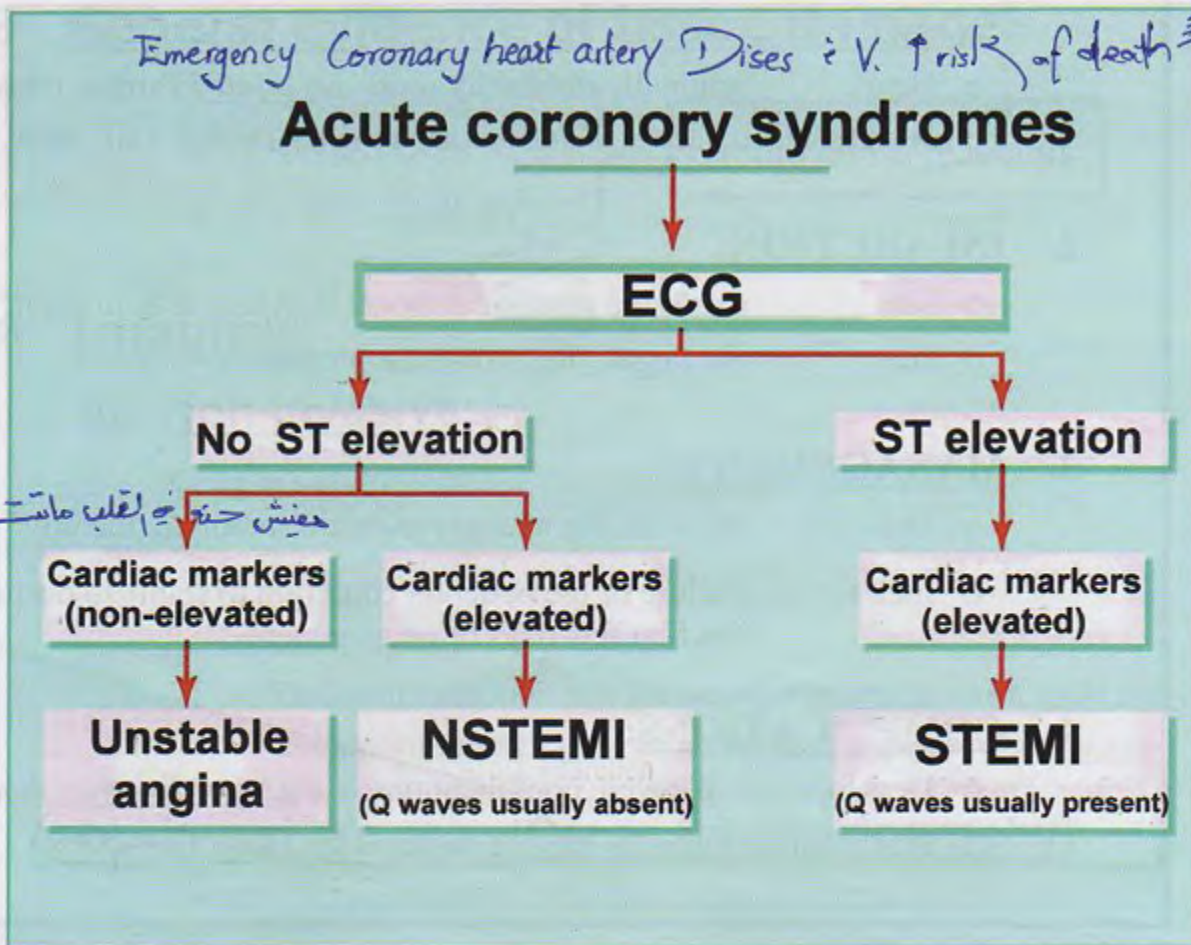
2. AMI:

- STEMI

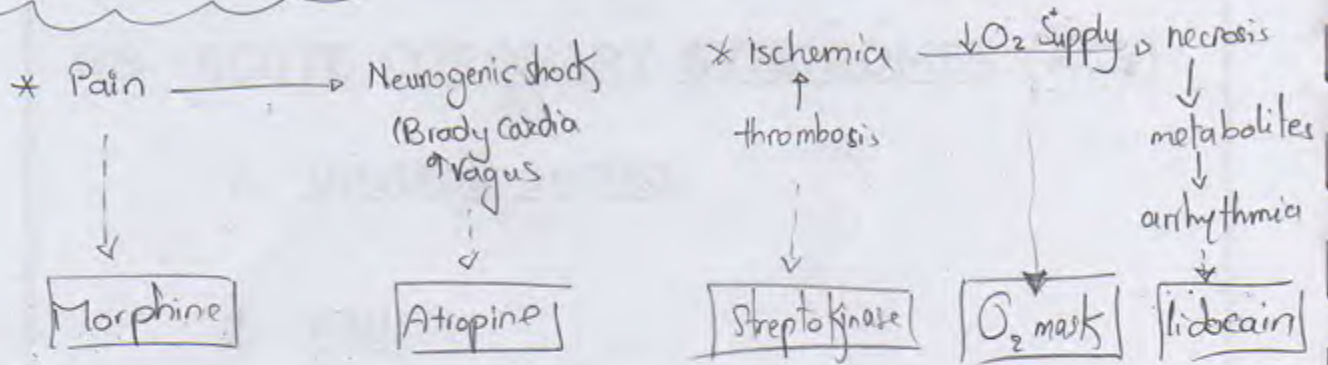
[confirmed by cardiac markers (enzymes)]

- NSTEMI

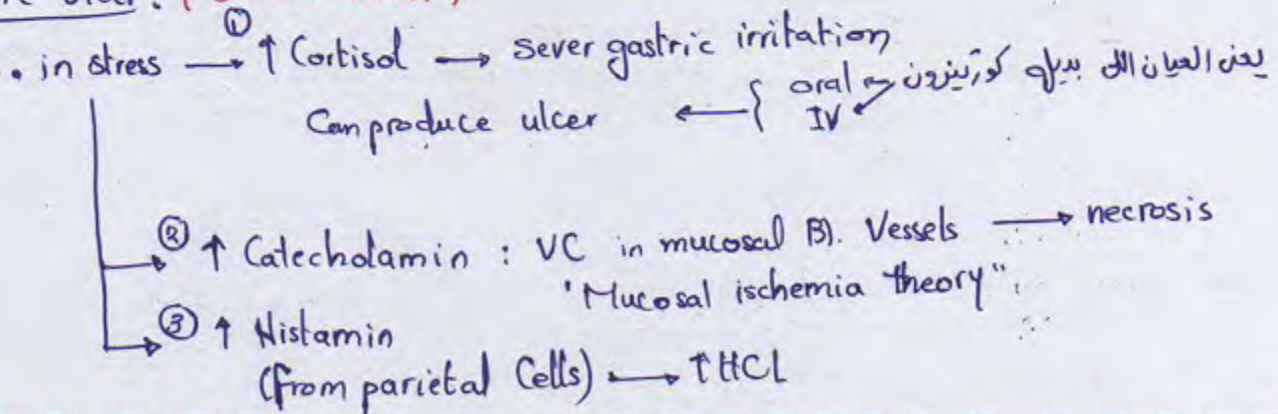
[diagnosed by cardiac markers (enzymes)]



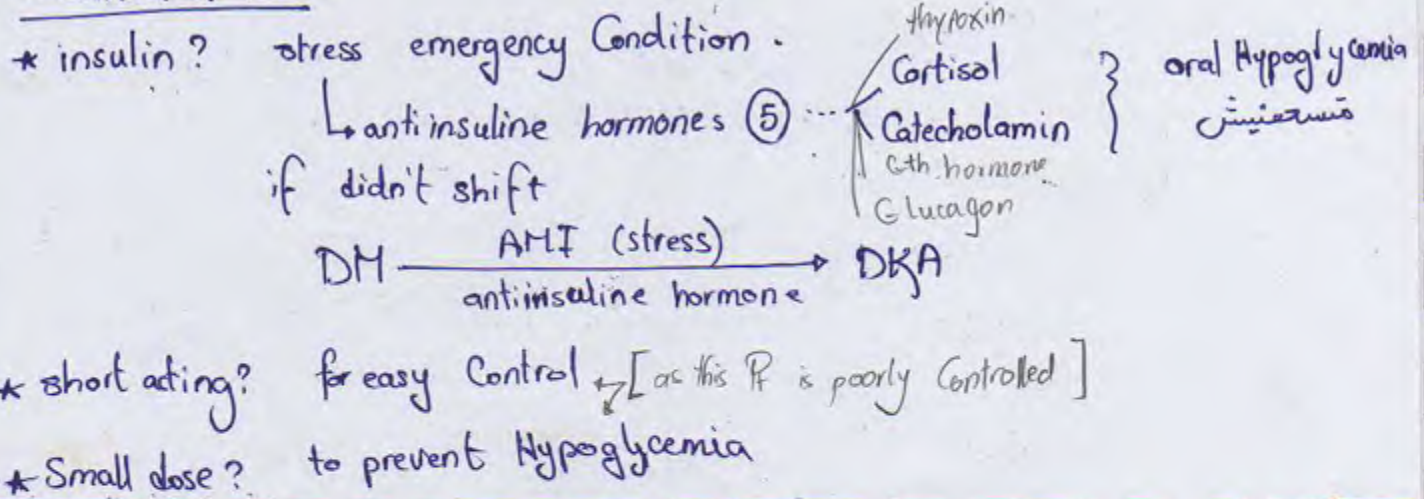
Pie hospital Phase



Peptic Ulcer: (stress ulcer)

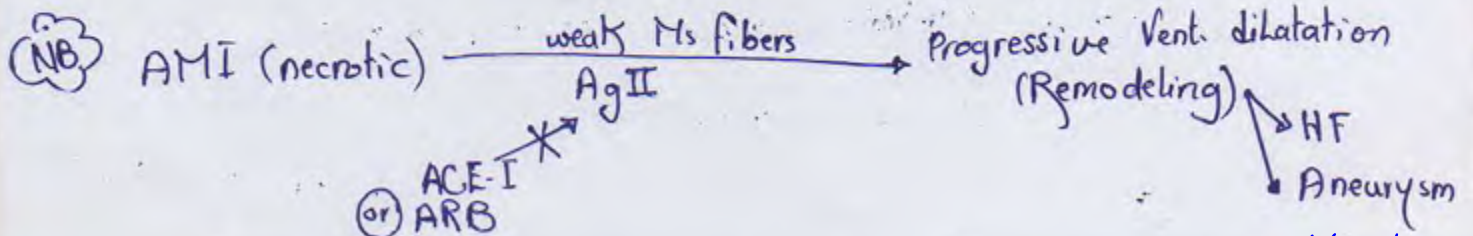


in Control of DM:



NB ACE-I indications in Cardio:

- ① HF
- ② Post MI
- ③ HTN



TREATMENT

1 PRE - HOSPITAL PHASE: في عربة الإسعاف

- a) Narcotic analgesics for pain: severe → Neurogenic shock
 - Morphine: 10 mg IV or pethidine: 50 mg IV.

ECG monitor

- b) Prophylaxis & Treatment of malignant ventricular arrhythmias:
 - Lidocaine: 100 mg IV. (to prevent VT → VF) حذر من ارتفاع ضغط الدم

shock & HT

- c) Treatment of increased vagal tone (causing bradycardia):

- Atropine: 1 mg IV.
 - If bradycardia persists → pacing is needed. (artificial pace maker) → ↑ HR
 زيادة معدل ضربات القلب

الحقن اذوية فورية

- d) Prehospital coronary thrombolysis: (streptokinase)

- Important for early protection of the myocardium.

NB one of causes of sudden death is occlusion of 1st main coronary artery

احتمال حدوث الموت المفاجئ

- e) Oxygen inhalation. (↑ O₂ supply) [IHD]

2 HOSPITAL PHASE:

"CCU"

1. General:

- Monitoring: continuous hemodynamic & ECG monitoring. Bl. Pr., Pulse
 - Management of urgent complications, e.g., "Catastrophic; early Comp."

other types of arrhythmia can give; Lidocaine

1. Fatal Ventricular arrhythmias: DC cardioversion.

2. Heart block: Pacing. (urgent)

- Rest: Physical, Mental (sedatives, e.g. Diazepam 2 - 5 mg tds).

- Diet: Light frequent meals & stool softeners to avoid constipation.

- Oxygen inhalation.

- Control of associated DM: "Silent MI"

- o Diet control. (strict Control) خفض السكر في الدم
 o Drugs: INSULIN (small doses of short acting).
 o AVOID: HYPOGLYCEMIA. ↑↑ size of MI : تأخرها في علاجها
 ↳ avoid Valsalva
 ↳ avoid situational syncope

- Prophylaxis against STRESS ULCER: (↓ HCL)

الأسيد

- Much more potent
 o Proton pump inhibitors (e.g. Omeprazole), or,
 o H₂ receptor antagonists (e.g. Ranitidine).

2. Refractory or continuous chest pain:

- ② ○ IV morphine or pethidine. (2nd injection) (مفيش الثالث: جيلو إدمان)
- ③ ○ IV Nitroglycerine (10 – 200 μg / min). → ↑ Supply: VD Coronary
↓ work: ↓ preload
- ④ ○ IV or oral β -blockers. → ↓ Cardiac work
- ① ○ OXYGEN INHALATION.

3. Angiotensin converting enzyme inhibitors: ACE-I

- They should be given within 24 hours to all patients post – MI to:

- ① Reduce the progressive ventricular dilatation (Remodeling) ^{due to weak H₂}
- ② Reduce the incidence & severity of HF. (early, catastrophic Complic.)

○ Can Give ARB ← [لا تفسد]

4. Reperfusion: (coronary thrombolysis) "to limit infarct size"

- ① ○ Fibrinolytic drugs: e.g. Streptokinase, 1.5 million IU (IV infusion).

• TPA

- ② ○ Timing: as soon as possible; maximum benefit occurs within: 4 – 6 hours after the onset.

(ممكن تديه حق لغايه ٤-٦ ساعة كي التا بوجع اقل والناتج احسن)

- ③ ○ Result:
 - immediate dissolution of the thrombus, and, saving of the myocardium. (limit infarction size)
 - if no infarction → no pathological Q in ECG

- ④ ○ After Fibrinolytic drugs:

to avoid Rebound thrombosis after fibrinolytic withdrawal { Antipaletelet (aspirin) & Anticoagulant (Heparin), are given with & after fibrinolytic drugs. (Maintain Anticoagulation State)

- ⑤ ○ Complications:

- Bleeding tendency.
- Reperfusion arrhythmias (e.g. AIVR). * (P200) تفزع فتجبن

Indications:

- Only in: STEMI.
- Not in: NSTEMI (no benefit from thrombolytic therapy in NSTEMI).

Contraindications:

- Bleeding disorders.
- Severe Hypertension. $> \frac{180}{110} \Rightarrow$ Cerebral Hge
- Hepatic failure. (already bleeding tendency)
- Active peptic ulcer. (Bleeding, perforating)
- Aortic dissection. (weak wall → rupture → bleeding & no thrombus to block the defect)

* AIVR = Accelerated Idio – Ventricular Rhythm

5. Drugs that reduce the work of the heart:

A) NITRATES: (IV nitroglycerine) ↓ Preload

- Given in the first days following AMI.

B) β - BLOCKERS: ↓HR, ↓BP, ↓Contractility

- Given in the first days following AMI.

Never CCB: as in MI → potent -ve inotropic

6. Anticoagulants:

① • Following thrombolytic therapy. (streptokinase) [to avoid rebound thrombosis]

② • Thrombo-embolic complications. (تكوين قيعات التخثرية مفرطة)

③ • Intracardiac thrombi detected by echocardiography. (Mural thrombus)

7. Antiplatelet drugs:

• **Aspirin** (low dose): 75 – 300 mg / day (single dose).

• **Dipyridamole**: 75 mg twice daily.

• **Ticlopidine**: 250 mg twice daily.

المعتدل / المنخفض • **Clopidogrel**: 75 mg once daily.

8. Mechanical revascularization:

Methods:

- PCI: (PTCA + Stent).
- Surgery: (CABG).

Indications:

① - In the patient who does not respond to thrombolytic therapy

② - Presence of a contraindication to thrombolytic therapy.

③ - Following thrombolytic therapy. → after thrombolysis still raw area for thrombosis

9. Treatment of early complications:

• Pump failure & shock: IV inotropics Dopamin & Doputamine

• Arrhythmias. (Lidocaine)

• Others: "Surgical ttt" e.g.

(rupture): Septal defect: closure of the defect.

3 **POST - HOSPITAL PHASE:** "After Discharge"

① - Treatment of late complications: بعد أسبوعين

- Myocardial aneurysm: aneurysmectomy.
- Frozen shoulder syndrome: corticosteroids & physiotherapy.

② - REHABILITATION:

- Gradual return to normal activity.
- Regular exercise.

③ - Control of risk factors of atherosclerosis:

e.g. DM, HTN, Smoking, etc... dyslipidemia

حتى من القلب مات
دبغته القلب مهدد

④ - Decrease the risk of post-infarction angina & re-infarction:

- ASPIRIN. ($\ominus$ platelet aggregation) [2yr prophylaxis]
- β -blockers. ($\downarrow$ HR, $\downarrow$ BP, $\downarrow$ Contractility) [لومديتوش جرعة في حق العيان] (β $\leftrightarrow$ CAD)
- STATINS. (treat dyslipidemia $\rightarrow$ treat of atherosclerosis)

هيميشي على
ال 3 أدوية دول
طول عمره

التأثيره مع بعض
For life

مثلا مالهوموش
e.g. bronchospasm

NB Aspirin in Angina is 1yr prophylaxis (P₁₃₃)

NB HTN + CAD $\rightarrow$ Give β -blocker (Highly indicated in this case)

Diagnostic criteria of MI (WHO criteria)

1. **History:** Ischaemic chest pain > 20 minutes (Typical chest pain).
2. **ECG:** Changes in serial ECG tracings (Typical ECG).
3. **Enzymes:** $\uparrow$ Troponins, CK - MB, (Typical enzymes).

SYSTEMIC HYPERTENSION

30-35% of population → leading Cause of death when complication occurs

DEFINITION

- Persistent elevation of the arterial blood pressure above 140/90 mm Hg in at least 2 measurements, on at least 2 separate occasions.

→ high normal

PREHYPERTENSION:

Recently, BP from 120/80 mmHg to 139/89 mmHg is defined as "prehypertension."

It identifies individuals at high risk of developing hypertension, (JNC 7)¹

Joint National Committee

WHITE COAT HYPERTENSION:

It is a phenomenon in which patients have elevated BP in a clinical setting But not when recorded at home.

It is believed that this is due to the anxiety some people experience during a clinic visit (Therefore: Ambulatory BP monitoring is required).

تربطه الجريان عشان
يقاس الضغط ٢٤ ساعة

SEVERITY

Systolic & diastolic → الاثنين هجين

الخطر بسبب ال complication

	Systolic	Diastolic
Normal	Up to 140	Up to 90
Hypertension		
Stage 1 (mild)	140 – 160	90 – 100
Stage 2 (moderate)	160 – 180	100 – 110
Stage 3 (severe)	More than 180	More than 110

نفسه كتر قرحه للأعلى
170 → moderate
95
155
115 → severe

TYPES

1. Isolated Systolic Hypertension: ISH (rise of SBP only)

- Increased LV stroke volume: Hyperdynamic circulation: e.g. AR.
- Decreased aortic compliance: Aortic atherosclerosis. (stiff = تصلب)

2. Systolic & Diastolic Hypertension: (rise of SBP & DBP)

- Essential (primary) hypertension.
- Secondary hypertension.

¹ JNC 7 (The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure)

② NB: Compliance = $\frac{\text{Change Vol}}{\text{Change Pr.}}$ i.e. $\uparrow \text{Pr.} = \frac{\text{Vol.}}{\text{Compliance}}$ → needed to accommodate Blood Vol. in Systole

→ Controllable

A) Essential (primary) Hypertension: (> 95 % of the cases)Age: It occurs in middle & old ages. (Never young age)**ETIOLOGY**

UNKNOWN

Predisposing factors:

1. Genetic: runs in families.
2. Obesity. ① atherosclerosis ② ↓ Physical activity ③ Insuline Resistance.
3. Stress.
4. Salt sensitivity: 60 % of hypertensive patients are salt sensitive.
5. Smoking.

مرتبطین
بعض
DM
HTN

T1DM

Insuline Resistance.

salt & water
retention
↓
G⁺-Na⁺
exchange

G⁺ entry to Vessel wall Ms

spasm: Vessels Contracts (VC)

از ای مش که
~~~~~

Theories for etiology: Pathogenesis

1. Activation of the **RAAS** (Hyper active: Renin)
  - ↑ secretion of renin which converts angiotensinogen to angiotensin I, and then to **Angiotensin II (AT II)** which causes hypertension through:
    - o AT II is a very potent VC causing ↑↑ PR.
    - o AT II stimulates aldosterone secretion → salt & water retention.

2. Activation of the **SNS**

- ↑ secretion of catecholamines will cause VC & ↑↑ PR.

## 3. Vascular hypertrophy: (&amp; endothelial dysfunction)

- ↑ production of certain **growth factors** → vascular hypertrophy (narrow lumen) → ↑↑ PR & hypertension.
- Hypertension → vascular endothelial injury (**endothelial dysfunction**) → further ↑↑ the production of growth factors → more hypertension.

Abnormal  
~~~~~

IGF
vicious
Circle
↑PR
↑HTN
injury

Mechanical
endothelial
damage

4. **INSULIN RESISTANCE: (IR)**

- Peripheral resistance to insulin occurs & → secondary **hyperinsulinemia** that elevates the BP through:

T2DM

- a) Increased Na reabsorption in the renal tubules. (aldosterone like)
- b) Increased sympathetic activity.

B) Secondary Hypertension:

③ (< 5 % of the cases)

- ① Cause – related hypertension.
- ② Curable hypertension.
- ③ Any age (including young)

NB Fat Cells, Liver Cells, Ms Cells are the most common insulin target

White Knight Love

Essential (1ry) HTN

- ① Unknown Etiology
- ② Controllable
- ③ Age: Middle & old
- ④ >95% of Cases
- ⑤ Predisposing Factors & theories :-

* Predisposing Factors:

- i - Genetic
- ii - Obesity
- iii - stress
- iv - smoking
- v - salt sensitivity 60%

* Theories of Etiology:

I RAAS

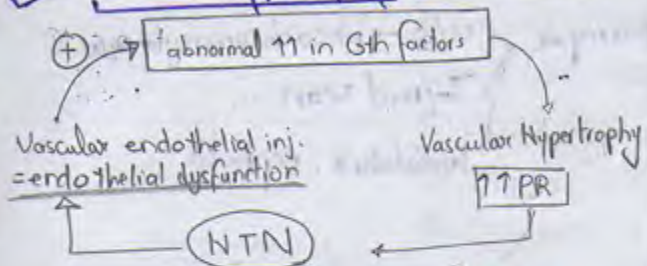
Hyper active system $\rightarrow$ $\uparrow$ Renin $\rightarrow$ A II

- ① $\uparrow$ pre load : ④ old : salt & water retention
- ② $\uparrow$ afterload : Sever VC in arteries

II JNS $\uparrow$ Catecholamines $\rightarrow$ VC

$\uparrow$ PR, $\uparrow$ after Load

III Vascular Hypertrophy & endothelial dysfunt.



IV Insuline Resistance $\rightarrow$ T₂DM

Peripheral resistance to insuline
= insuline insensitivity
= 2ry Hyper insulinemia

aldosterone Like
($\uparrow$ Na⁺ reabsorption)

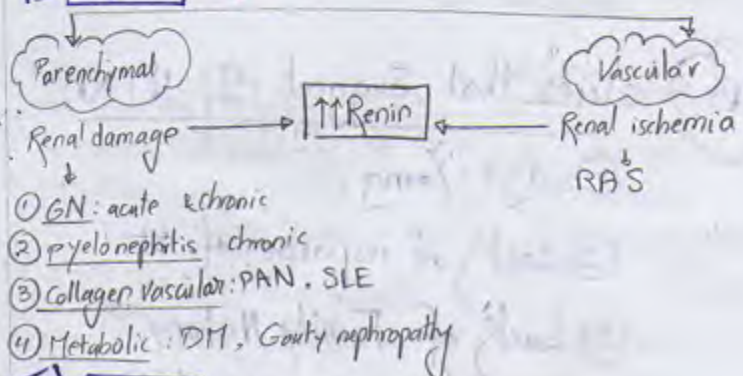
$\oplus$ Sympathetic

Secondary HTN

- ① Cause Related
- ② Curable
- ③ Age: Any (occur in Young)
- ④ <5% of Cases
- ⑤ Etiology:

when you treat the Cause there'll be no need
for anti hypertensive drugs.
 $\rightarrow$ Just Symptomatic tht

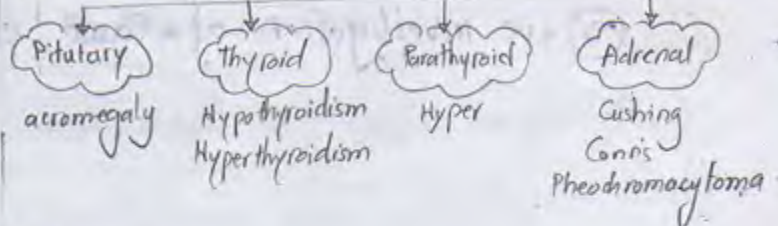
I Renal $\star$ الكلى



II Neuro $\oplus$ VMH in medulla

- ① TICT
- ② lesion in medullar or Hypothalamus (imitative lesion)

III Endocrinal



IV Drugs

- ① Catecholamines
- ② Corticosteroids
- ③ CCPs (contraceptive pills)
- ④ Cyclosporine
- ⑤ Carbenoxolone

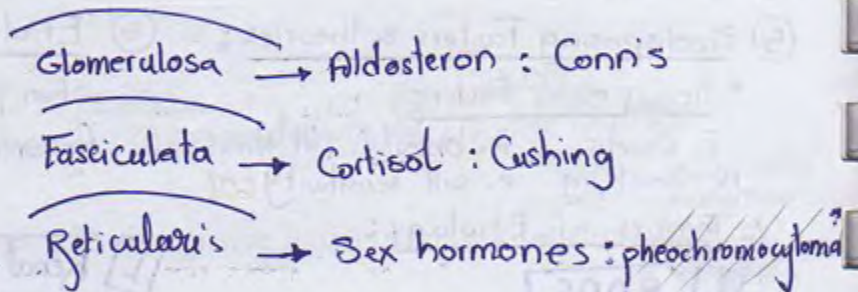
V Miscellaneous

- ① Coarctation of Aorta
- ② Preeclampsia & eclampsia
- ③ PAN
- ④ PRV

NB SLE & PAN:

- ① GN "↑↑ Renin"
- ② Vasculitis "small vessels" → end arteritis obliterans → ↑ PR → HTN
↓
Renal ischemia
- ③ ttt is by Corticosteroids

NB Adrenal Gland



NB Clues that Suggest 2^o HTN

- ① Age: Young
- ② Lack of response of ttt
- ③ Lack of Family History
- ④ Presence of C/P of a Cause : example
 - ← Moon face
 - ← acromegaly
 - ← audible bruit : RAS
 - ← Puffiness of eyelid (nephritis, myxedema)
- ⑤ +ve investigation of a Cause : example
 - ← U/s → pheochromocytoma
 - ← Thyroid scan
 - ← Hematuria : nephritis

ETIOLOGY

1. Renal causes:

a) Renal parenchymal: → Renal damage → ↑↑ Renin

- ① GN: Chronic & acute. → nephritic
- ② Pyelonephritis: Chronic pyelonephritis. (Never acute) Chronic = Persistent damage & fibrosis
- ③ Collagen diseases: SLE & PAN. (uric acid)
- ④ Metabolic: Diabetic nephropathy & Gouty nephropathy.

b) Renal vascular:

- Renal artery stenosis. → Renal ischemia → ↑↑ Renin

2. Endocrinal causes:

- head → • Pituitary: Acromegaly. [↑ Gth Hormone → Vascular Hypertrophy → ↑ PR]
- neck → • Thyroid: Hyperthyroidism, ^{SBP only} or Hypothyroidism.
- Parathyroid: Hyperparathyroidism. → ↑ cholesterol
- abdominal → • Adrenal gland:
 - ↳ ↑ Ca²⁺ → VC
 - ↳ Mucopolysaccharoids in Vessel wall. → Hypertrophy
- Adrenal medulla: Pheochromocytoma.
- Adrenal cortex: Cushing's syndrome & Conn's syndrome.

3. Neurological causes:

- ↑ ICT. (Brain tumor, abscess, edema; → irritate) → VMC in medulla → sympathetic → VC
- Lesions of: medulla & hypothalamus.

4. Drugs:

- Catecholamines. (ttx: Bronchial asthma)
- Corticosteroids. (as Cushing)
- Cyclosporine. (immuno suppressive 3 in organ transplantation)
- CCPs.
- Carbenoxolone. (antacid in peptic ulcer: affect Na⁺ ...)

5. Miscellaneous causes:

- Coarctation of the aorta. → HTN in upper half of the body
- Toxemia of pregnancy.
- Polyarteritis nodosa. → Vasculitis
- Polycythemia rubra vera. (مضطرب RBCs) ↑ Viscosity → HTN

- Important question: "Self - assessment"

"Mention the clues that suggest Secondary Hypertension".

(NB) Gout: skin, kidney, joints

(NB) ↑ Bl. Pr. in lower half: AR

CLINICAL PICTURE

3 Types of Clinical Presentations

Silent Killer
مرض من غير
سبب!
مرض من غير عرض!
الضغط مش احساس
الضغط دا يا خياش

1 Benign Hypertension:

Years
→ duration before Complications

- A mild form of HTN that runs a slow, long & usually asymptomatic course.

Symptoms

- Asymptomatic: القاعدة إنه ميستركش "the majority of patients"
- Headache, tinnitus, dizziness, palpitation & epistaxis: "a minority of patients" قلة

Signs

1. General:

- Pulse: ↑ pulse volume in: ISH.
- Blood pressure: persistent elevation above: 140/90.
- Fundus examination: reveals signs of HTN retinopathy.

2. Cardiac:

a) Precordial examination:

- Signs of LVH: with heaving apex.

b) Auscultation:

- S2: accentuated.
- Systolic ejection click.
- Systolic ejection murmur: due to relative AS.
- Soft early diastolic murmur: due to functional AR.
- S4: (strong atrial contraction) over the mitral area.

P42
الضغط
win as pulm HTN

2 Hypertensive urgency:

→ weeks, months to reach complications

- It is a severe form of HTN with rapid rise of BP: > 220 / 120 mm Hg.
- It is NOT ASSOCIATED with: target organ damage.

3 Hypertensive emergency:

- It is a severe form of HTN with rapid rise of BP: > 220 / 120 mm Hg.

- It is ASSOCIATED with: target organ damage.

- The organs primarily affected include:

- CVS: Acute pulmonary oedema, AMI, Dissecting aortic aneurysm.
- CNS: Hypertensive encephalopathy, Cerebral hemorrhage & SAH.
- KIDNEY: Acute renal failure.

- There are 2 types:

- Accelerated HTN: associated with retinopathy (hemorrhages & exudates). Stage III
- Malignant HTN: associated with retinopathy (PAPILLEDEMA). Stage IV

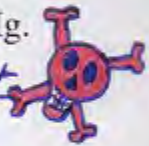
acute LSHF

plaque rupture

Mechanical

Massive
Brain edema

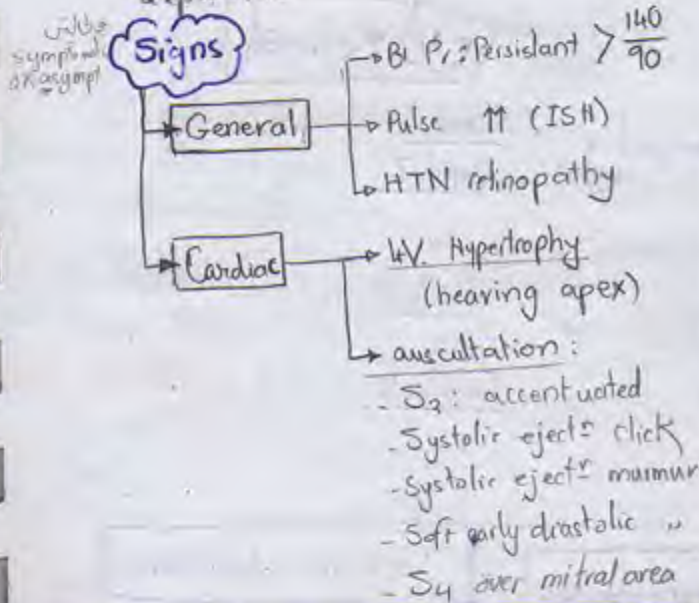
Much more Common than



Fundus Exam $\leftarrow$ HTN: retinopathy
 Inf. endo Carditis: Roth spots
 AR: Backer

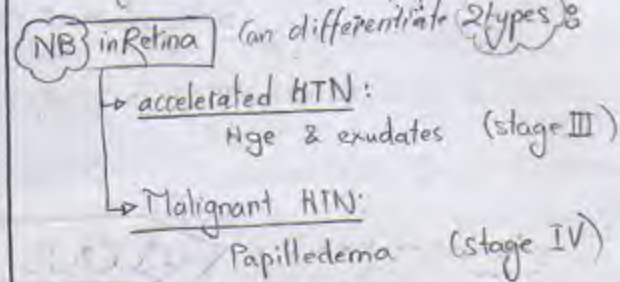
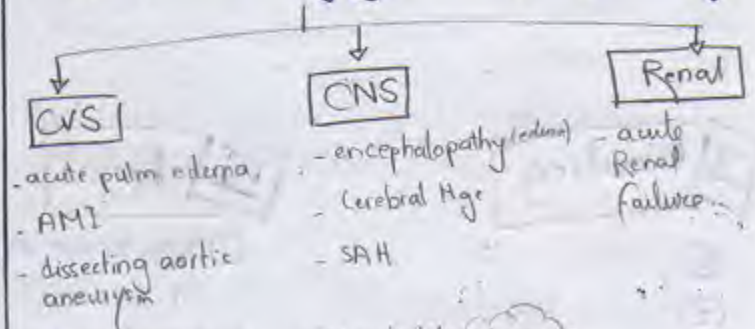
Benign HTN

- ① Mild form
- ② slow Course
Duration; may take years to develop complications
- ③ Persistent elevation above $\frac{140}{90}$
- ④ usually: asymptomatic
Cases & symptoms shows Headach, tinnitus, dizziness, palpitation & epistaxis



Hypertensive "Emergency / Urgency"

- ① Severe form
- ② Rapid $\uparrow$ in blood pressure weeks, months to reach complications
- ③ Rapid rise $> \frac{220}{120}$
- ④ in Hypertensive urgency $\rightarrow$ No target organ ϕ
 in " Emergency: ϕ target organ damage.



try Benign	sp. situations	Urgency	Emergency
stepped Care therapy step I: ACE-I step II: ACE-I + $\beta\beta$ step III: ACE-I + $\beta\beta$ + α CCB step IV: step III + Hydralazine The starting step: mild HTN $\rightarrow$ step I moderate " $\rightarrow$ step II Severe " $\rightarrow$ III	IHD: ✓: A, B, C X: VD HF: ✓: A, D X: B, C RF: ✓: α -methyl dopa Hydralazine X: K ⁺ sparing diuretic Bilateral RAS: X: ACE-I, ARB BA: X $\beta\beta$ DN: ✓: A, C X: B, D Pregnancy ✓: α -methyl dopa Hydralazine X: A, D	↓↓ Bl. Pr. within 24 hrs. to avoid & stop complications • Rapid $\downarrow$ b/b is avoided • Not below 100 diastolic or will $\downarrow$ perfusion • $\beta\beta$ + CCB + Diuretic Triple therapy Hydralazine Na nitroprusside Cause if worse 2ry after tit of the emergency case must treat the Cause	① Hospitalization: ICU ② $\downarrow$ BP $\rightarrow$ 100 diastolic within 1hr - Diazoxide 300mg IV - Hydralazine 20mg IV - Na nitroprusside 0.5-10 μ g/kg/min IV - Furosemide 40mg IV - α -methyl dopa 250mg IV best in APE ③ After Control $\rightarrow$ oral antihypertensive starting from step III ④ specific $\uparrow$ for each emergency e.g. HTN encephalopathy: Mannitol + anticonvulsant

NB 2ry: treatment of the Cause

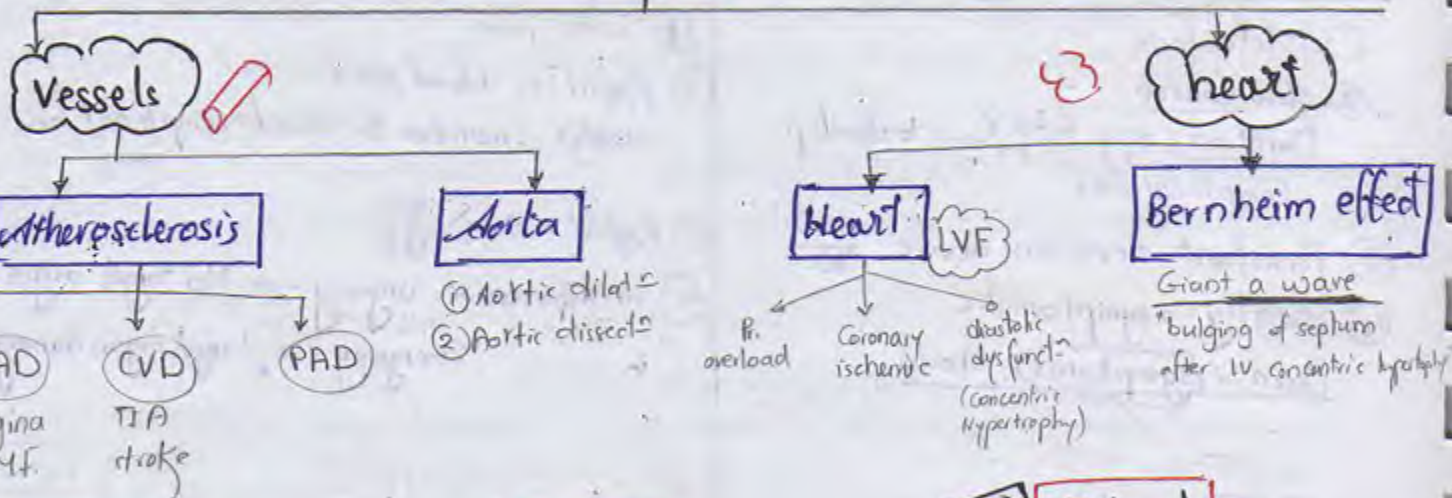
NB if worse 2ry after tit of the emergency case must treat the Cause

WhiteKnightLove

Small Vessels $\Rightarrow$ Eye, Kidney, Nerves

Complications of HTN

I CVS

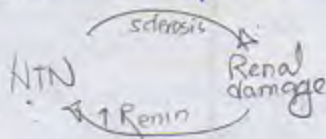


II Bleeding

- Epistaxis
- Cerebral Hge
- hemoptysis, hematemesis

III Renal

- Hypertensive nephropathy
RF due to nephrosclerosis
- Hematuria & proteinuria



IV Retinal

- Grade I:** Narrow retinal a.
- Grade II:** Sclerosis of retinal a. (silver wiring), kinking of veins on arterial crossing
- Grade III:** Grade II + flame shape Hge, fluffy cotton exudate
- Grade IV:** Grade III + papilledema

V CNS

Atherosclerosis

TIA, stroke

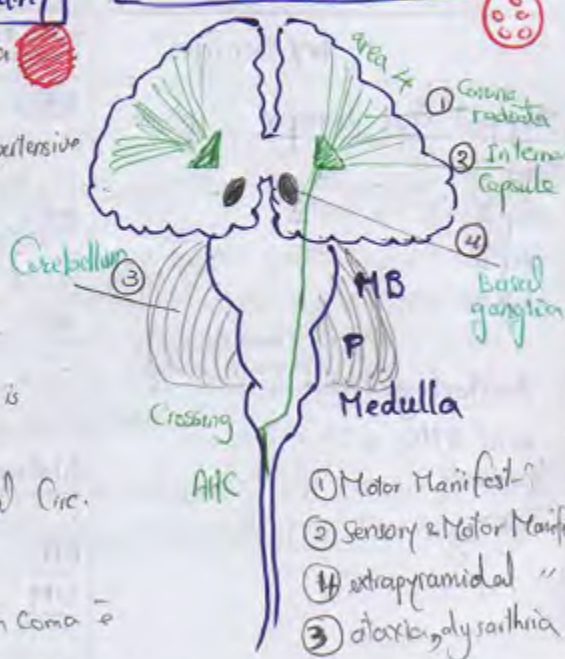
Hge

Cerebral Hge, SAH, signs of lateralization

Hypertensive encephalopathy

- Sudden massive Brain edema
- Sudden severe HTBP due to:
 - sudden stoppage of anti-hypertensive drug (Rebound)
 - Sudden severe stress
 - ichthyotic
- Generalized VC occurs but Vasomotor tone of Cerebral aa. is weaker than the peripheral so $\rightarrow$ blood is pooled in Cerebral Circ.
- C/P:
 - $\uparrow$ ICT
 - Drowsiness then Coma $\rightarrow$ Convulsions
 - No signs of lateralization

Lacunar infarctions



- Motor Manifestation
- Sensory & Motor Manifestation
- extrapyramidal
- ataxia, dysarthria

COMPLICATIONS

1. CVS:

1. ATHEROSCLEROSIS: which may lead to, (affect Big Vessels)
 Mechanical endothelial dysfunction (injury) →
 • CAD (Angina & AMI).
 • CVD (TIA & Stroke).
 • PAD. → Peripheral ischemia (عرج الجراحي)

2. HEART: LVF, due to,
 ① Pressure overload, ② coronary ischemia, ③ diastolic dysfunction.

3. AORTA: Aortic dilatation & Aortic dissection.
 → Hypertrophy → ↓ Compliance (stiff.)
 → injury to intima (تلف)

4. BERNHEIM EFFECT:

- Concentric hypertrophy of the LV may cause bulging of the septum in the RV cavity, leading to slight impairment of filling, and giant a wave in the neck veins.



2. CNS:

1. CEREBRAL ATHEROSCLEROSIS: (TIA & Stroke).

2. HEMORRHAGE: Signs of lateralization (Cerebral hemorrhage & SAH).

3. HYPERTENSIVE ENCEPHALOPATHY: "Sudden Severe Brain Edema"

Mechanism:

أحيى لا انا بقاى!

"Never stop anti Hypertensive drug: Rebound HTN"

- Attack of sudden severe ↑↑ of BP with generalized arteriolar spasm.
- Since the vasomotor tone of the cerebral arteries is weaker than that of the peripheral arteries, therefore the blood will rush to the cerebral circulation & causes cerebral oedema. (transudation; massive)

→ Sudden Severe Stress idiopathic

Clinical Picture:

- Manifestations of ↑↑ ICT: headache, vomiting, blurring of vision. (Projectile → Papilledema)
- DCL - Disturbed consciousness: Drowsiness then **coma** with: (inhibition of Cortical neurons due to edema)
- Convulsions.
- **NO SIGNS OF LATERALIZATION.**

4. Lacunar infarctions:

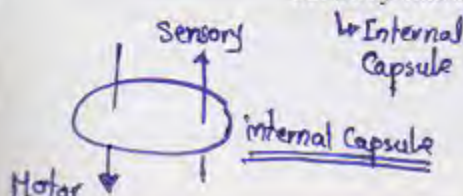
→ en2 → liquefactive necrosis → lacunae (فوهات)

- Multiple small infarcts usually occurring in the corona radiata, internal capsule, cerebellum or basal ganglia.
- Clinically, they may be asymptomatic, or present with mild motor or sensory manifestations, ataxia, dysarthria or extrapyramidal manifestations.

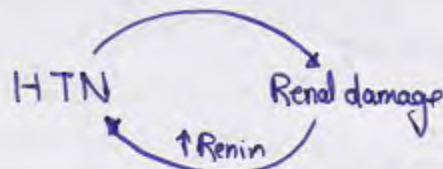
→ Corona radiata
→ int. capsule

→ Cerebellum

→ Basal ganglia



NB CT scan differentiate between edema & bleed



HTN
Mechanical end. damage
sclerosis in vessels of kidney

3. Renal:

- Hypertensive nephropathy: renal failure due to nephrosclerosis.
- Hematuria & proteinuria. (damage in glomerular ~~damage~~ vessels)

4. Retinopathy: (4 grades)

- Grade I: Narrowing of the retinal arteries.
- Grade II: Marked sclerosis of the retinal arteries (silver wiring), Kinking of the veins at the arteriovenous crossings,
Plus: grade I.

accelerated HTN - Grade III: Flame shaped hemorrhages & fluffy cotton exudates ...
Plus: grade II.

Malignant HTN - Grade IV: Papilledema
Plus: grade III.

5. Bleeding:

- Epistaxis: warning sign / ↓ Bl. P. the most common.
- Cerebral hemorrhage: the most serious.
- Other bleeding: (e.g. hematemesis or hemoptysis): ... uncommon.
ميجملو ش تقریباً
محصولات لا يبقی فی کما ن
local

INVESTIGATIONS

1. For the diagnosis of the cause: (in secondary HTN)

1. Renal investigations: urine, renal functions, renal imaging.
2. Endocrinal investigations: lab tests for hormones, imaging.
3. Neurological investigations: CT, MRI brain.
4. Blood: CBC. (polycythemia rubra vera)

2. For the diagnosis of complications:

1. Cardiac investigations: CXR, ECG, Echocardiography.
2. Neurological investigations: CT, MRI brain.
3. Renal investigations: urine, renal functions, renal imaging.

3. For detection of associated risk factors of atherosclerosis.

TREATMENT

1 Aim of treatment

- Complications: To avoid or ↓ the development of complications.
- BP goal:
 - To achieve & maintain BP < 140 / 90 mm Hg in hypertensive patients.
 - To achieve & maintain BP < 130 / 80 mm Hg in hypertensive patients with DM.

(2 risk factors in atherosclerosis)

2 Plan of treatment

A) Primary (essential) Hypertension:

- I. Non- pharmacological treatment.
- II. Pharmacological treatment:
 1. For Benign Hypertension.
 2. For Hypertensive urgency.
 3. For Hypertensive emergency.

B) Secondary Hypertension:

- Treatment of the cause.

I. Non-Pharmacological Treatment (Life style modification)

a) Dietary:

- Salt: must be restricted.
- WEIGHT REDUCTION IN OBESE PATIENTS.

b) Non-dietary:

- Smoking: must be stopped.
- REGULAR PHYSICAL ACTIVITY.

II. Pharmacological Treatment

1. FOR BENIGN HYPERTENSION (Stepped Care Therapy)

- ▶ The ttt passes in steps, if one step is not sufficient, we proceed to the next step:
- **Step I:** ACE – I, or β -blocker or Calcium blocker, or Diuretic.
 - **Step II:** Combinations of 2 drugs of step I.
 - **Step III:** Combinations of 3 drugs of step I.
 - **Step IV:** Add α -adrenergic blocker or Hydralazine to step III drugs.
- ▶ The starting step:
- In mild hypertension: start by step I.
 - In moderate hypertension: start by step II.
 - In severe hypertension: start by step III.

2. FOR HYPERTENSIVE URGENCY

- ▶ Aim of ttt is to lower the BP rapidly (within 24 hours) to stop complications. However, too rapid $\downarrow$ of BP should be avoided as it may $\rightarrow$ organ hypoperfusion. (ie not below 100 diastolic)
- ▶ TTT is urgently started using: diuretic, BB, VD, (triple therapy) $\leftarrow$ oral IV

3. FOR HYPERTENSIVE EMERGENCY

▶ General treatment for all emergencies

1. Hospitalization in an ICU.
2. Rapid reduction of the BP to approximately 100 mm Hg diastolic: within one hour using rapidly acting drugs, e.g. $\leftarrow$ v. potent IV
 - Diazoxide: 300 mg rapidly IV.
 - Hydralazine: 20 mg IV. (Best in ARF) $\rightarrow$ $\uparrow$ Renal bl. supply
 - Na nitroprusside: 0.5 – 10 μ g/kg/min IV (best in APE).
 - Frusemide: 40 mg IV (best in APE).
 - α -methyl dopa: 250 mg IV. (Best in ARF) $\rightarrow$ $\uparrow$ Renal Bl. supply
3. After control of BP, oral antihypertensive treatment should be started with drugs in step III. (Severe HTN)

▶ Specific treatment for each emergency (Symptomatic ttt to target organ damage)

- A) Hypertensive Encephalopathy: $\rightarrow$ Osmotic diuresis
- Dehydrating measures: concentrated mannitol or glucose.
 - Anticonvulsants.
- B) Other emergencies: refer to the related chapters.

Anti-hypertensive drugs

Group	Drug	Mode of action	Dose	Side effects
1. Diuretics	Refer to the chapter of "Heart Failure"			
2. Anti-adrenergics				
a) Centrally acting	Alpha-methyl dopa (aldomet)	Central inhibition of the sympathetic system	250 - 1000 mg tds orally	- CAH <i>chronic active hepatitis</i> - Hemolytic anemia
	Clonidine	Central inhibition of the sympathetic system	0.1 - 0.6 mg bid orally	- Postural hypotension - Rebound hypertension
b) Ganglion blockers	Trimethaphan	It blocks impulse transmission in the autonomic ganglia	1 - 6 mg / min IV	- Postural hypotension - Constipation & urinary retention
c) Nerve ending blockers <i>(obsolete)</i>	Guanethedine	It blocks the release of catecholamines from the adrenergic neurons	10 - 100 mg / day orally	- Postural hypotension
	Reserpine	It depletes the stores of catecholamines at the nerve endings	0.1 - 0.2 mg tds orally	- Depression
d) Alpha blockers	Prazocin	It blocks the alpha receptors causing VD	1 - 10 mg bid orally	- Sudden syncope - Tachycardia
e) Beta blockers	- Propranolol - Atenolol	- Reduction of CO	Refer to the chapter of "angina"	
f) Combined alpha & Beta blockers	Carvedilol	It blocks both alpha and Beta receptors	25 - 50 mg / day orally	- Same as β - blockers
3. Vasodilators <i>Direct = smooth muscle relaxants</i> <i>Emergency</i>	a) Hydralazine	Direct smooth muscle VD	10 - 50 mg tds orally or IV	- Tachycardia - Precipitates angina - Lupus-like syndrome <i>skin rash, arthralgia, pleurisy, pericarditis</i>
	b) Minoxidil	Direct smooth muscle VD	2.5 - 50 mg / day orally	- Tachycardia - Precipitates angina - Hirsutism
	c) Sodium nitroprusside	Direct smooth muscle VD	0.5-10 $\mu\text{g/kg/min}$ IV	- <i>فوق الحد</i> - Thiocyanate poisoning <i>متدرج شدة الأعراض</i>
	d) Diazoxide	Direct smooth muscle VD	300 mg IV rapidly	- Hyperglycemia - Hyperuricemia

4. Angiotensin converting enzyme inhibitors (ACE - I)	Captopril	It blocks the conversion of Ag I to Ag II	Captopril: 12.5- 50 mg tds orally	Dry cough Proteinuria ! idiosyncrasy !! Pancytopenia OBM Hyperkalemia Renal failure if given in bilateral RAS
	Ramipril		Ramipril: 5- 10 mg/day	
5. Angiotensin receptor blockers (ARBs)	Losartan	It competitively Inhibits the binding of Ag II to its receptors	50 mg once daily orally	Hyperkalemia Renal failure if given in bilateral RAS
6. Calcium channel blockers (CCBs)	Amlodipine	It blocks the entry of Ca into the cells causing vasodilatation	5 - 10 mg / d orally	Refer to the chapter of "angina "
	Diltiazem		30 - 90 mg tds orally	
	Nifedipine		10 - 30 mg tds orally	
	Verapamil		80 - 160 mg tds orally	

شفاویہ دوائیں

Anti - Hypertensive drugs in certain situations

Clinical situation	Preferred drug	Contraindicated drug
A) Cardiovascular		
1. IHD	- CCBs (angina) - BB (angina & post-MI) - ACE - I (Post-MI)	- Hydralazine - Minoxidil ↳ severe tachy → ppt angina
2. Heart failure	- Diuretics - ACE - I	- CCBs - BB → حقیقتاً : ½ اثر ہے
B) Renal		
1. Renal failure	- Alpha-methyl dopa ↑Renal Blood Flow - Hydralazine	- Potassium-sparing diuretics حیوانی K ⁺ سے
2. Bilateral RAS		- ACE - I, ARBs
C) Others		
1. Bronchial asthma		- BB → selective 100% مفید نہیں ہے
2. Diabetes mellitus	- ACE - I, ARBs, CCBs ↳ nephropathy → potent arteriodilator	- BB, Diuretics ↳ impotence افضل حدیثوں ↳ Mask Hyperglycemia (CSF)
3. Pregnancy	- Alpha-methyl dopa - Hydralazine	- Diuretics. teratogenic - ACE - I.

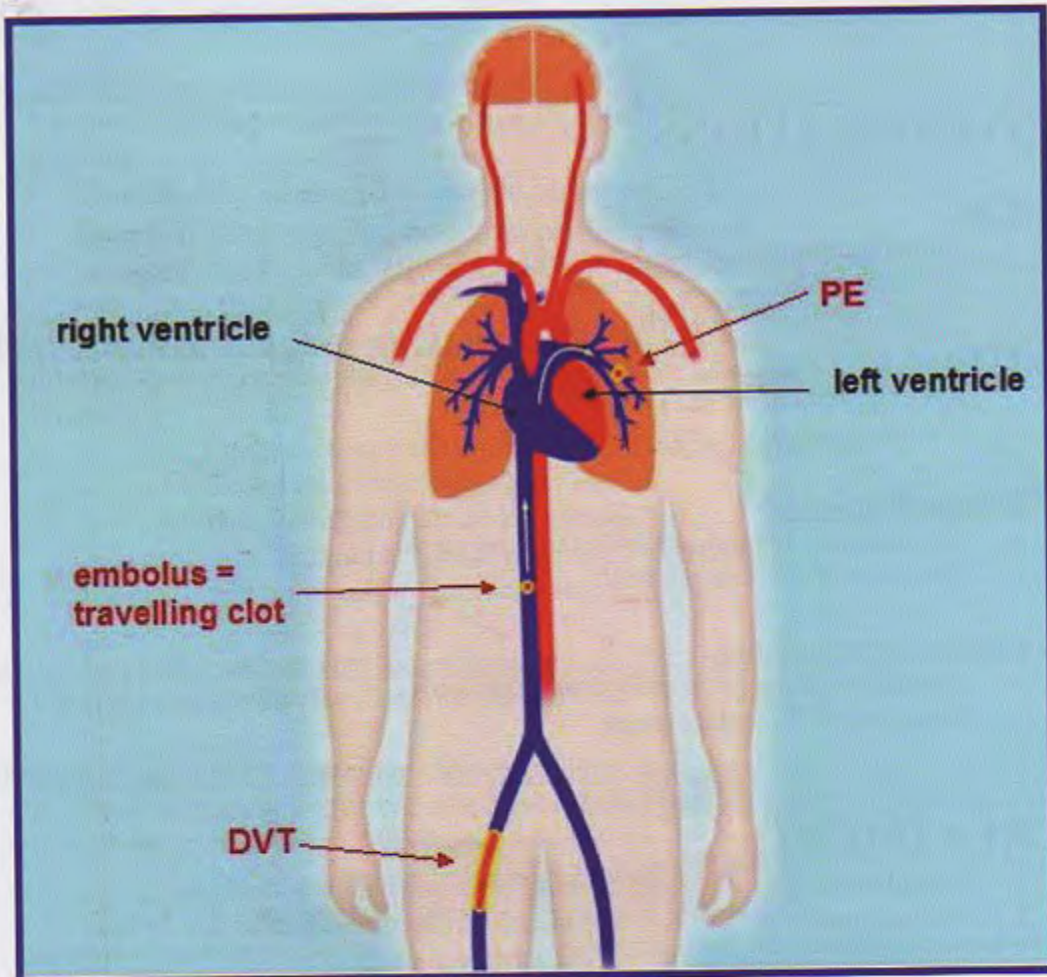
Important question: "Self - assessment"

"What is meant by: Refractory Hypertension" ??

PULMONARY EMBOLISM

DEFINITION

- Sudden occlusion of a lung artery, usually due to a blood clot that travelled to the lung from a distant source in the circulation.
- Therefore, pulmonary embolism is not considered a disease; it is considered: "A complication of an existing thrombosis, most commonly DVT of the leg".



ETIOLOGY

"Source of emboli"

1 Peripheral veins

A) Blood emboli: (DVT) (90 %), due to (Virchow's triad), *

1. Abnormalities in blood flow: "Slow circulation, Stasis"
 - Prolonged bed rest.
 - Heart failure.
 - Varicose veins.
2. Abnormalities in blood composition: "Hypercoagulability"
 - Refer to "Blood". DIC, PRV
3. Abnormalities in vessel wall: "Endothelial injury"
 - Trauma.
 - Inflammation. (Behcet's disease) (Venulitis)

B) Other (unusual) emboli: (very rare)

1. Fat emboli. (Fracture bone, liposuction, surgery)
2. Air emboli. (iatrogenic, during insertion of CVP catheter)
3. Parasitic emboli. (eg. ascaris)
4. Malignant cell emboli.

2 Right side of the heart (10 %)

1. RA thrombi: in cases of AF.
2. RV thrombi: in cases of RV infarction.
3. Right sided vegetations: in cases of infective endocarditis. (IV drug abusers)

3 Paradoxical embolus (very rare)

- Embolus from the left side of the heart passes through a defect:
e.g. VSD or ASD to the right side of the heart.

* The German doctor **Rudolf Virchow** (1821-1902), described the triad.

Etiology Pulmonary Embolism

I Peripheral Vein

- ① Bleed emboli (DVT) 90%
- ② others (Rare)

"Virchow's Triad"

Hypercoagulability

↓ Anti: Thrombin III

↓ Pro S. ↓ Pro C

↓ α₂ Macroglobulin

↓ α₁ antitrypsin

↓ heparin cofactor

+ DIC

PRV

• Heparin

Endothelial injury

↓ Inflammation

Behcet's disease, Vasculitis

trauma

Pelvic operation

Varicose Veins

Compression by tumour, pregnancy

CHF (Congestion → VUR)

Prolonged recumbency (bed rest, long trips)

Parasitic

Malignant cell

AF

II Rt side of the heart

- ① Rt atrial thrombi
- ③ Rt Vent. thrombi
- ③ Rt sided Vegetations

10%

III Paradoxical Embolus

Very rare
- Emboli from Lt side
Passed through a defect
VSD, ASD to Rt side



eg DVT

Features of the source of embolus

C/P P.E

Pneumonia, Lung abscess

- ① infected
- ② Number

Features of P.E.

Size

Medium Sized

Pulm infarction (<60% occlusion)

Minute
↓ may be
asymptomatic
↓
Repeated showers
↓
thrombo embolic pulm NTN
↓
RVF
↓
Subacute Cor pulmonale

General
* Fever, Jaundice
* tachypnea
* tachycardia
may be the only presenting feature

Pleural (opposite infarct)
* Pain: ↑ insp. stitching
* Rub. rough
* effusion (complication)

Lung
* dyspnea
* Cough
* hemoptysis

DR chest pain (acute)
dyspnea (acute)
hemoptysis

Pulm infarction (>60% occlusion)

Big

Acute Massive P.E. (60-80%)

Sudden death >80% (main pulm trunk)

- ① Chest Pain (sudden severe retrosternal)
- ② acute dyspnea (may be cyanosis)
- ③ RVF (rapid development of pulm HTN)
- ④ shock (cardiogenic)

DD

- ① Massive P.E
- ② Arrhythmia (VT)
- ③ lt main coronary occlusion
- ④ Rupture Cardiac aneurysm
- ⑤ MVP
- ⑥ AS

DD Massive HJ
- Massive lung collapse
- Tension pneumothorax

Not red tender swollen calf

CLINICAL PICTURE

I. Features of the source of embolism, e.g. DVT. tender Hot, swollen Calf M's tense

II. Features of pulmonary embolism vary according to:

- ① o Whether the embolus is infected or not:
 - Infected embolus may lead to pneumonia & lung abscess.
- ② o Number of emboli. → many vessels
- ③ o Size of embolus. → large one'll close big vessel → more severe case

A) Minute embolus

1. Asymptomatic. (in case of single embolus → minute ineffective infarction)
2. Repeated showers of minute emboli cause occlusion of large number of pulmonary arterioles → thrombo-embolic pulmonary hypertension → RVH
→ subacute cor pulmonale. VC قفلة في الأوعية RVH RVF
→ need time for repeated showers to be enough to cause pulm HTN & RVF.

B) Medium-sized embolus → large vessels occluded → pulmonary infarction (< 60 % occlusion)

- Clinical picture:

1. General affection:

- Fever & jaundice may occur. necrosis → pyogens Hemolysis → dead area Hemoptysis من الدم Sensative But not specific
- Tachypnea & Tachycardia could be the only presenting features. → Hypoxia & RC → Compensatory to low CO

2. Pleural affection: (pleurisy)

- Symptom: sudden stitching chest pain which ↑ by inspiration & cough. (stretch of pleura) "pleuritic Pain"
- Sign: there is pleural rub.
- Complication: pleural effusion may develop.

3. Lung affection: Acute dyspnea, cough & hemoptysis.

- Differential Diagnosis:

- From other causes of: acute chest pain. e.g. pericarditis, infarct
- From other causes of: acute dyspnea. e.g. Bronchial asthma
- From other causes of: hemoptysis. e.g. TB

إدارة
management
20-25 marks
add D.D.
وكل كطين عن 10 إلى 20

C) **Big embolus**

1. **Acute massive pulmonary embolism:** (60 – 80 % occlusion)

- **Clinical picture:**

- الإصابة
- ↓ ↓ ↓ ↓
- ischemic pain due to low CO (typical anginal pain)
- | | | |
|---|--------------------------|--|
| ① | Acute Chest pain: | sudden severe retrosternal similar to that of AMI. |
| ② | Acute Dyspnea: | and may be cyanosis. (Hypoxia) |
| ③ | Acute RVF: | rapid development of PH & acute RVF. → acute Cor pulmonale |
| ④ | Shock: | cardiogenic. |
- فشل القلب ويعتوا

- **Differential Diagnosis:**

From other causes of: acute ¹chest pain, acute ²dyspnea, acute ³RVF & ⁴shock.

Left Right مع ال

① Acute Massive P.E.

- | | |
|---|---|
| ② | • Massive myocardial infarction. |
| ③ | • Massive lung collapse. |
| ④ | • Tension pneumothorax. |

2. **Sudden death.**

(> 80 % occlusion)

eg. embolism in Main pulmonary trunk

Did you know ...

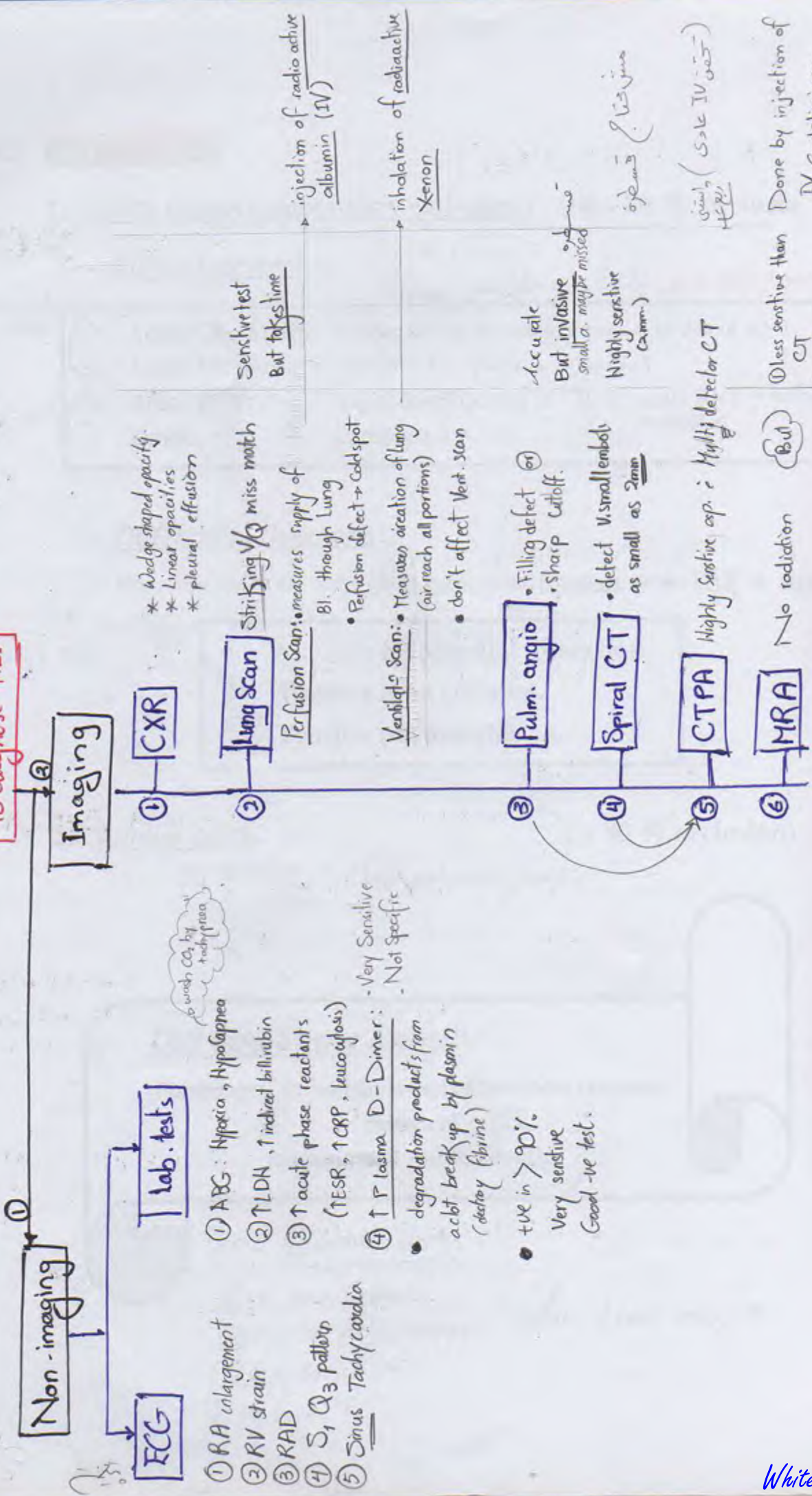
Pulmonary embolism is one of the most common causes of unexplained sudden deaths

Causes of Sudden death :-

- ① Lt main Coronary
- ② Rupture aortic aneurysm: Rupture of heart aneurysm
- ③ Arrhythmia (VF)
- ④ Massive PE
- ⑤ AS
- ⑥ MVP

Investigations P.E. ^① to diagnose DVT _{e.g duplex u/s}

to diagnose P.E.



INVESTIGATIONS

I. INVESTIGATIONS for diagnosis of DVT

"the Cause"

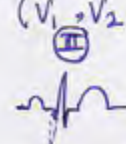
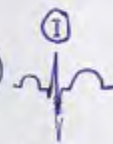
e.g. Duplex ultrasound. *accurate, sensitive, non invasive*

II. NON - IMAGING INVESTIGATIONS

1) ECG:

- ① RA enlargement
- ② RV strain. (evidence of pr. overload : V. non-specific)
- ③ RAD. (Right axis deviation)
- ④ S1 Q3 pattern. (deep S in I & deep Q in III)
- ⑤ SINUS TACHYCARDIA.

(P - pulmonale). tall & peaked
depressed ST segment & inverted T (V₁, V₂, V₃)
according to direction of electricity in Bulk of Muscle



كابو عشان كذا :
كده بيطلع طرفة
هشوف ساعلي
ايه
تقطع على
ساعة
متشخصش به
Sensitive الالاسين ال tachypnea
كاتب من الالاسين ال tachypnea

2) Laboratory tests:

- Arterial blood gases: Hypoxia & Hypocapnea.
tachypnea → wash CO₂
- Elevated serum LDH. (non specific)
- Elevated serum indirect bilirubin. (Hemolysis) (Jaundice)
- General evidence of tissue damage:
 - Leukocytosis.
 - Raised ESR.
 - Positive CRP.

Acute phase reactants

tissue inflam. tissue damage

• PLASMA D - DIMER: most imp. Lab

- It is a blood test that measures a substance in the blood that is released when a clot breaks up, i.e. it reflects the breakdown of fibrin by plasmin (endogenous thrombolysis). → By fibrinolytic system.
- It is positive in more than 90 % of patients, THEREFORE:
A normal test makes pulmonary embolism not likely. (Good -ve test)
- ★ Very sensitive test. (But) not specific

acute
Pulm. edema
P₉₁
"كافو افرصة : (التي في)
الاشارة
ساعة"

- ① CTPA & pulm. angiography
 ② MRA & CT ~~childhood~~ helical
 ③ Lung scanning V/Q ratio
- ترتيب الاربعة والثلثة

III. IMAGING INVESTIGATIONS

1) CXR

DD? D shaped opacity in CXR

- ① mesothelioma of pleura
 ② Pulm. infarction
 ③ encysted pleural effusion

- Wedge shaped opacity. (infarction)
- $\pm$ Linear opacity. $\rightarrow$ V. small infarction
- $\pm$ Pleural effusion. (Basal opacity rising laterally exudate)



Perfusion defect

= Normal Ventilation

لریم اناتو کر من الاشی
 لکن فی اوجان بشعور الانسین



2) Lung scanning

"Ventilation - perfusion scan"

- A test that uses a **radioactive material** to see if air and blood are flowing normally to areas of the lung.
- A lung ventilation/perfusion scan (V / Q scan) is actually **two tests** performed simultaneously:

• Perfusion scan:

- It is performed by injecting **radioactive albumin** into a vein.
- The lungs are then scanned to detect the location of the radioactive particles as blood flows through the lungs.
- It measures the supply of blood through the lungs.
- Pulmonary embolism appears as a perfusion defect (cold spot).

• Ventilation scan:

- It is performed by scanning the lungs while the patient inhales **radioactive gas** (Xenon).
- It measures the ability of air to reach all portions of the lungs.
- Pulmonary embolism does not affect the ventilation scan.

لکن بیاض و قوت

Pulmonary embolism causes striking V / Q mismatch

Sensitive Test

3) Pulmonary angiography

Femora V.

- Accurate, BUT invasive. (Catheter into pulm. a. $\leftarrow$ Rt heart $\leftarrow$)
- Positive results consist of: a filling defect or sharp cutoff of the affected artery caused by the embolus.
- Small artery may be missed

4) **Spiral (Helical) CT scan**

مش قسطره

- Highly sensitive.
- It can detect very small emboli (as small as 2 mm).

5) **CTPA**

(تحقق المادة IV عادي) واستقر مع C

- **C**omputed **T**omography **P**ulmonary **A**ngiography.
- Highly sensitive: especially with the recent **multidetector CT**.
high resolution

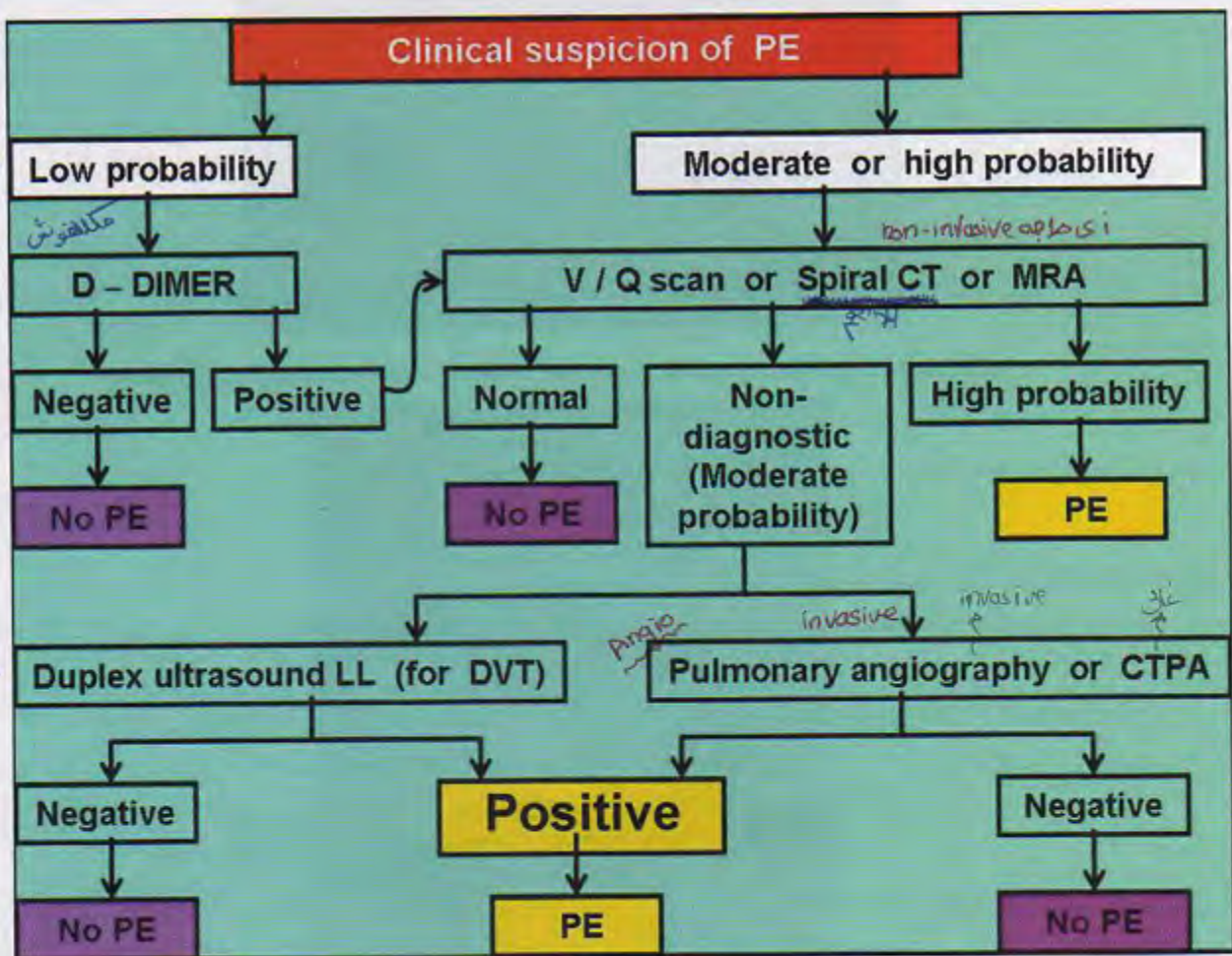
6) **MRA**

- It is performed following IV injection of gadolinium.

① No radiation

② less Sensitive than CT

③ Claustrophobia



Diagnostic algorithm for pulmonary embolism

TREATMENT

I. Prophylactic:

1. Prophylaxis & treatment: of the predisposing factors. HF, DVT
2. Prophylactic anticoagulation: in predisposed patients. Prolonged bed rest

II. Therapeutic:

1. Hospitalization in ICU. *oral*

2. GENERAL TREATMENT

a) Symptomatic treatment:

- morphine also RC de cat vi*
- Pain: Pethidine 50 mg IM, **but avoid morphine.** *Fatal mistake → RC*
 - Dyspnea: Oxygen inhalation.
 - RVF. *→ Ht of HF? →* *(E3)*
 - Shock. *→ +ve inotropes*

b) Treatment of the original source of embolism. *DVT till Anticoagulants → 6 months*

3. SPECIFIC TREATMENT

If the patient is hemodynamically unstable (Shock or HF) *Massive P.E.*

a) Thrombolytic therapy: "Streptokinase"

- 250,000 units IV bolus *followed by* 100,000 units / hour, *followed by* anticoagulation.

CTPA as CT spiral

b) Pulmonary embolectomy: *elective surgery*

- In the patient who **does not respond** to thrombolytic therapy.
- Presence of a **contraindication** to thrombolytic therapy.

If the patient is hemodynamically stable

Streptokinase

Further thrombosis & embolization are prevented by:

a) Anticoagulation:

- o Heparin IV for: **5 days**.
- o Heparin & oral anticoagulants, e.g. warfarin for: **5 days**.
- o Warfarin for: **6-3 months** at least.

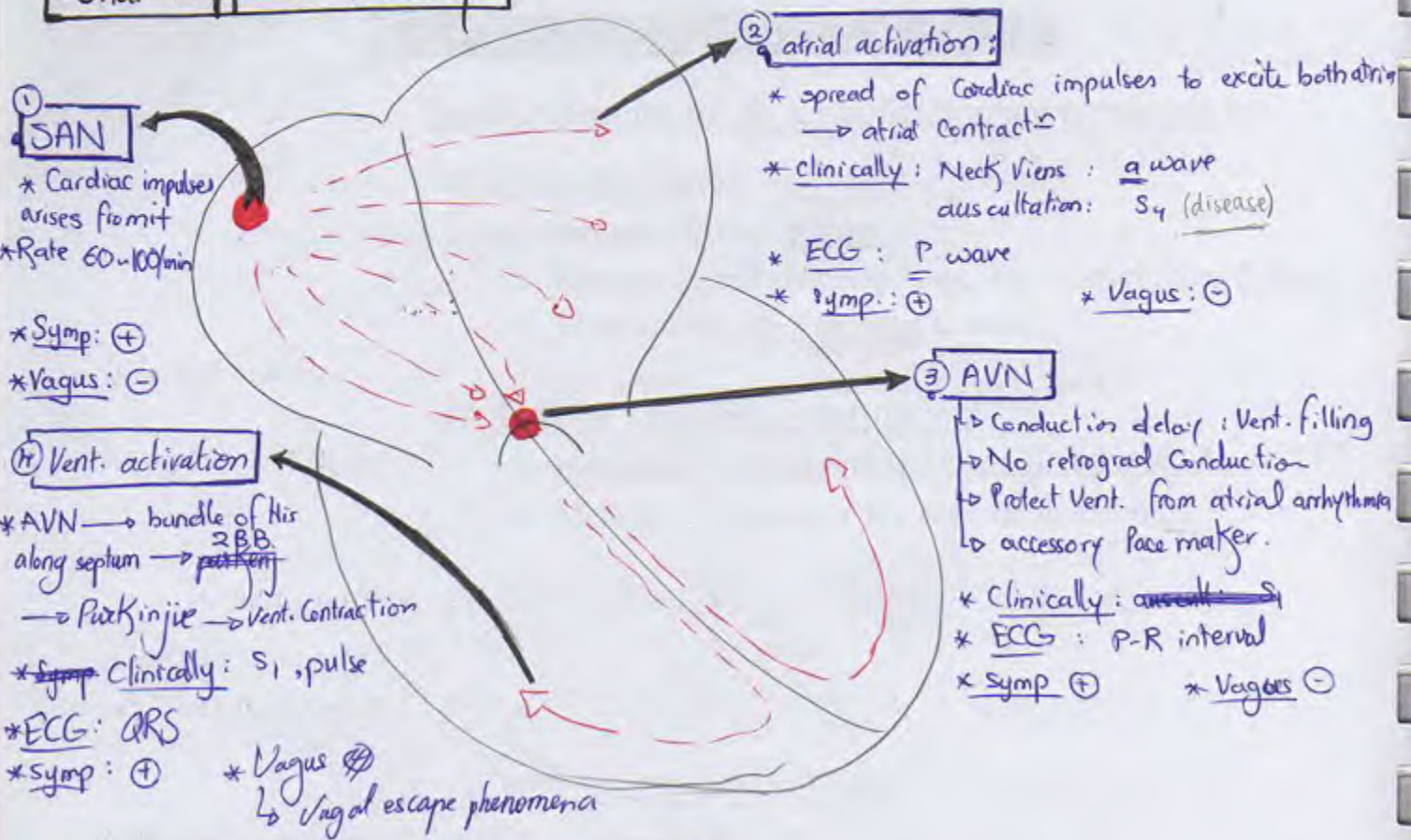
b) Inferior vena cava interruption:

- o Indication: contraindication to anticoagulants. / recurrent P.E
- o Method: Insertion of a filter or an umbrella.

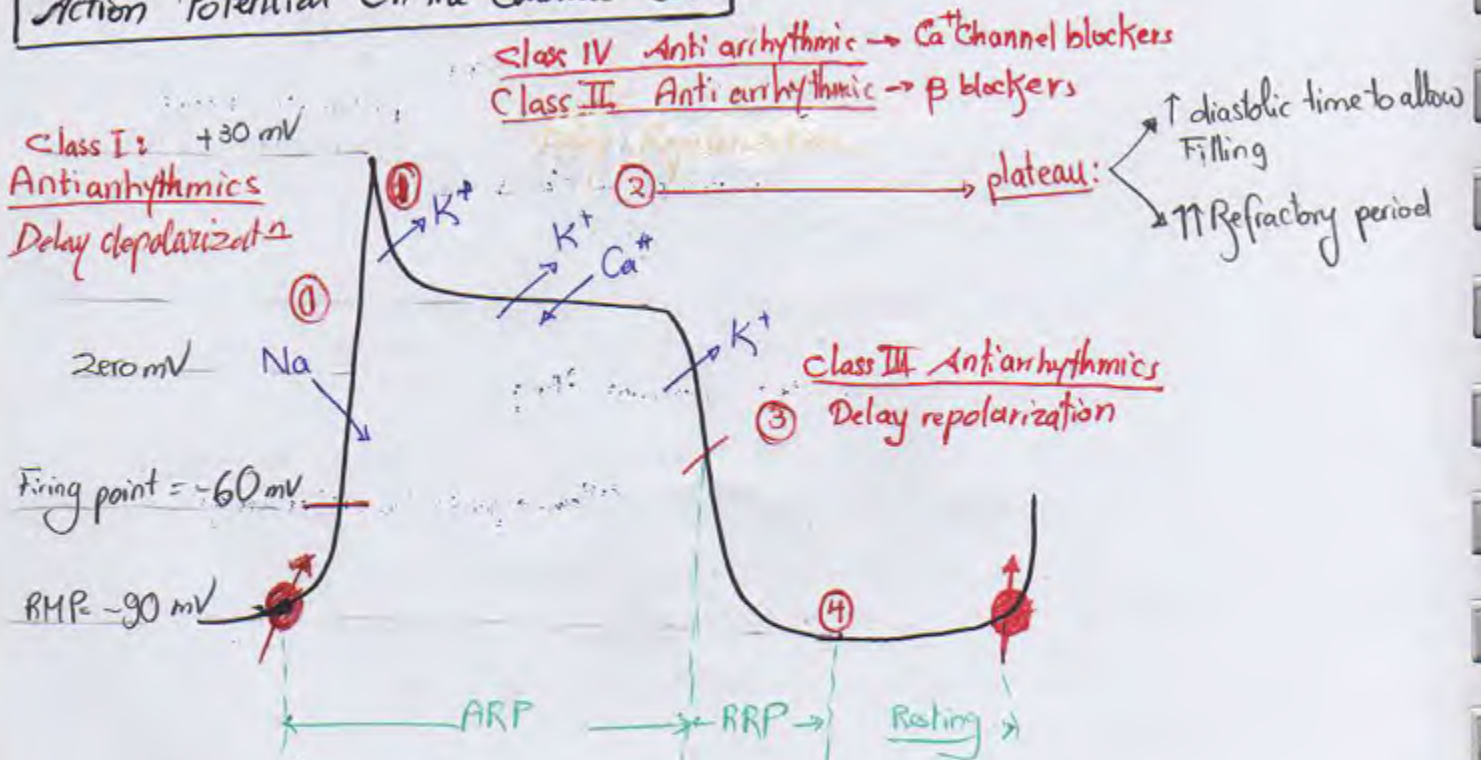
Arrhythmias

abnormal change in
 Rate
 Rhythm

What happens Normally?



Action Potential On the Cardiac Cell

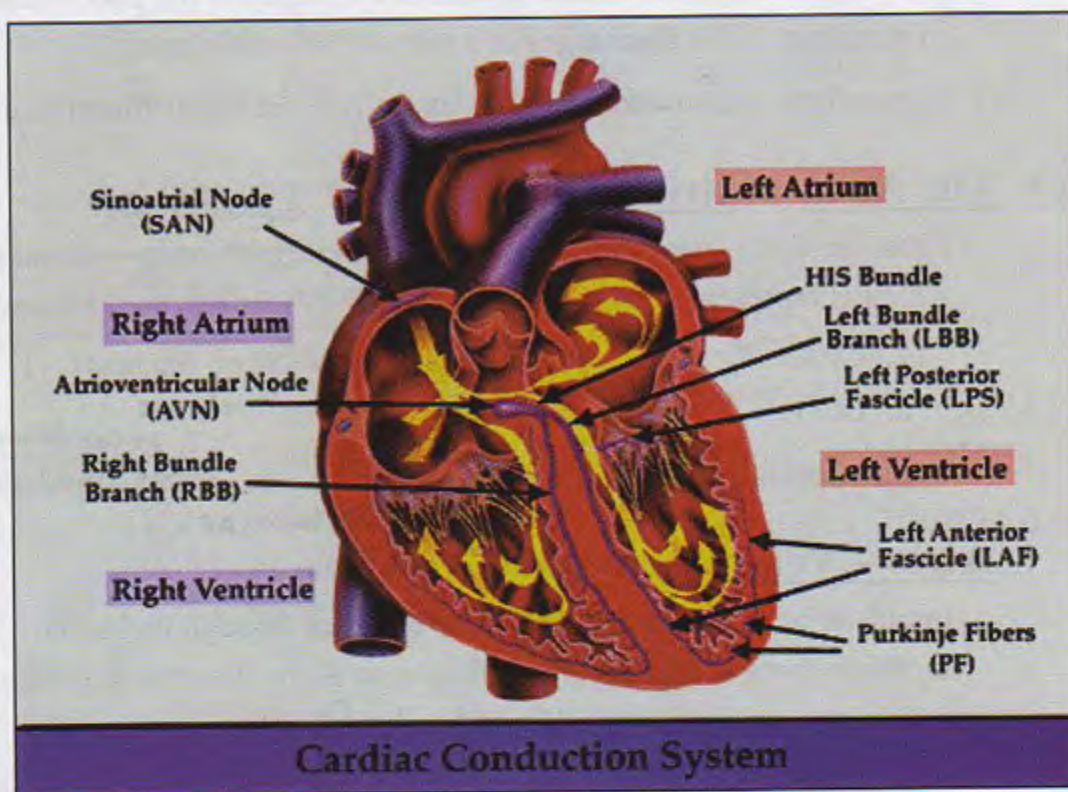


• The Ventricles (ventricular activation):

- Impulses pass from the AVN to the Bundle of His in the inter-ventricular septum, then along the 2 bundle branches & finally Purkinje fibers to terminate in the ventricular myocardium → ventricular contraction which is represented by:

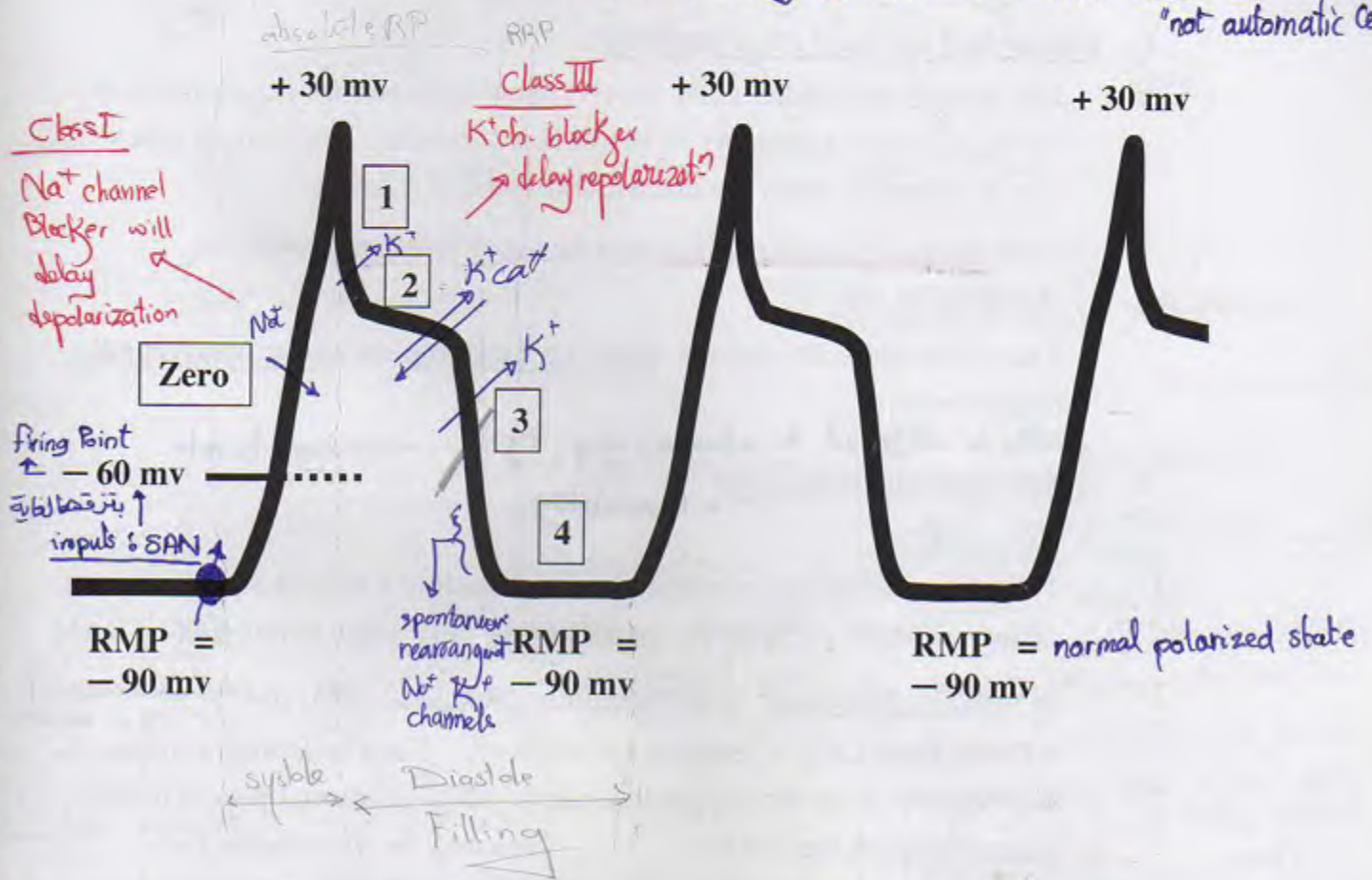
1. Clinically: radial pulse & first heart sound (S_1) [*Muscular Component*]
2. ECG: QRS complex.

- The ventricles are supplied by sympathetic fibers only (no vagal supply).
(*Vagal escape phenomena*)



Action potential of the cardiac cell

Electrical activity when $\oplus$ Normal Cardiac cell
"not automatic cells"



Phases of the action potential

- | | | | |
|------|---|--|---|
| Zero | = | rapid D epolarization | (entry of Na ⁺ inside the cell) |
| 1 | = | early rapid R epolarization | (exit of K ⁺ outside the cell) |
| 2 | = | P lateau | (exit of K ⁺ is equilibrated by Ca ⁺⁺ influx) |
| 3 | = | final rapid R epolarization | (exit of K ⁺ outside the cell) |
| 4 | = | RMP (R esting M embrane P otential) | |

plateau $\leftarrow$ $\uparrow$ Diastolic time allows filling
prolonge refractory period

Absolute Refractory P. = 0 + 1 + 2 + $\frac{1}{2} \times 3$
RRP = $\frac{1}{2} \times 3$

(NB) RMP in SAN = -60 $\rightarrow$ so automatically beating

What happens abnormally ??

(Mechanisms of arrhythmias)

(A) Disturbance of impulse formation:

1. Disturbed normal automaticity:

- The natural pacemaker cells (SAN) show spontaneous depolarization during diastole (phase 4) of the action potential, resulting in a new action potential when the electric threshold is reached.
- Such diastolic depolarization may be accelerated or slowed by: changing its rate.
- These changes will produce sinus tachycardia or sinus bradycardia, respectively.

• SAN is subjected to adrenergic or vagal ... → change its rate

2. Abnormal automaticity: = ↑ excitability

• Normally:

The normal working myocardial cells remain in a resting state at a high negative RMP (−90 mV), depolarizing only when stimulated by SAN.

- In certain diseases: (e.g. ischemia), (Manipulation, stretch, electrolyte disturbance, digitalis) RMP of these cells is reduced (−60 mV). Such cells may exhibit spontaneous diastolic depolarization & act as abnormal pace-makers, independent on the SAN;

This may be responsible for:

atrial or ventricular or nodal (junctional) arrhythmias.

↳ tachycardia

(السرعة من SAN)

(B) Disturbance of impulse conduction:

1. Decreased conduction:

- There is loss of the ability of the action potential to stimulate the myocardial cells, e.g. heart block.

2. Re-entry: *

- A wave of depolarization may be forced to travel in one direction around a ring of cardiac tissue.

- A circus movement will result producing a tachycardia,

e.g. paroxysmal tachycardias.

* Two conditions are required for re-entry to occur.

⇒ See Below →

The most common cause for tachycardia

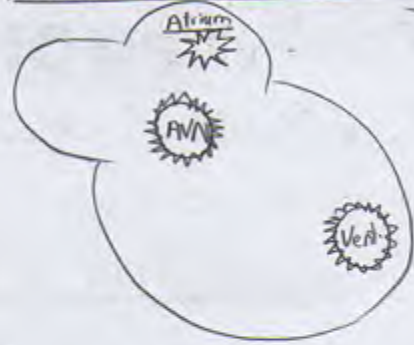
I Disturbance of impulse formation :

① Disturbed Normal automaticity



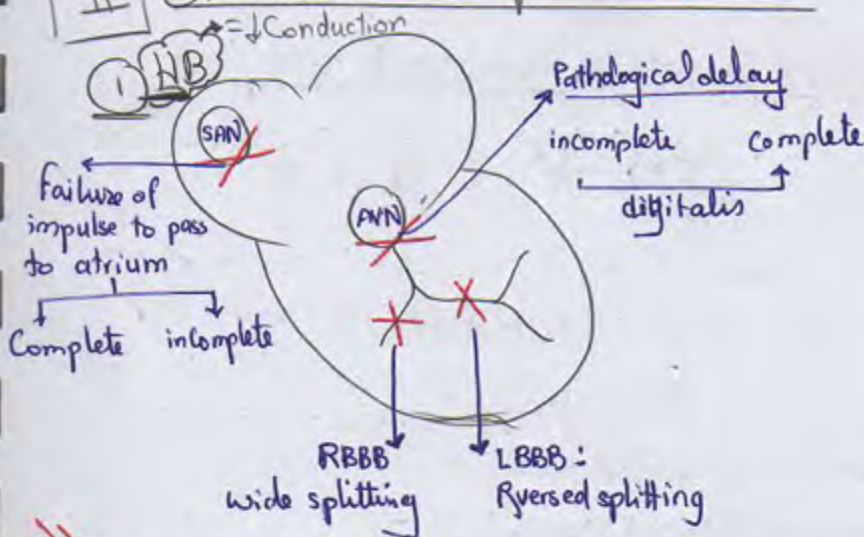
- SAN is still pacemaker
- Sinus tachy or brady

② Abnormal Automaticity



- SAN is not pacemaker
- Atrial, Nodal or Vent. arrhythmia

II Disturbance in impulse Conduction:-



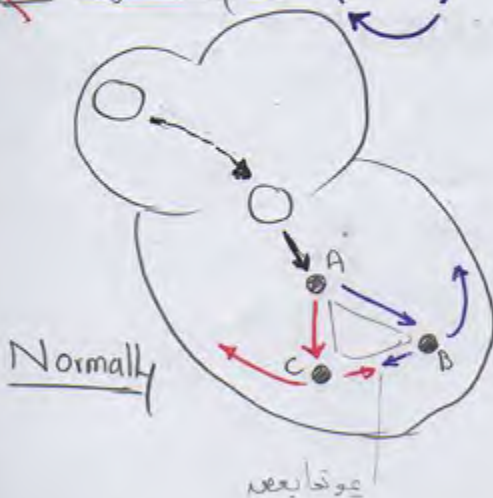
Lines of th?

- ① maintain antiarrhythmic drug
- ② th of Cause: e.g. ischemia

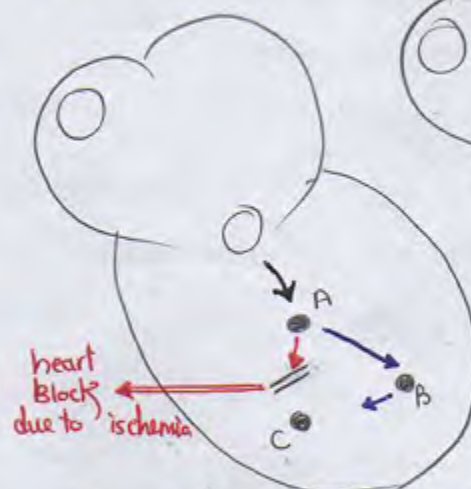
How to treat re-entry?

- ① TT Conduct?: in RP $\rightarrow$ $\downarrow$ conduction
- ② TT RP: Na^+ channel Blockers
- ③ DC shock: will depolarize all Ms $\rightarrow$ the circuit $\uparrow$ stop as it $\uparrow$ find all Ms in RP $\rightarrow$ cut circuit $\rightarrow$ stop the focus But $\uparrow$ re-occur (Paroxysmal)

② Re-entry :-



Normally



heart Block due to ischemia

- ① unidirectional Block in 1 fiber
(it's due to ischemia
it's not dead $\rightarrow$ not infarction)

- ② slower Conduction from the other side
(to reach point A in resting not in RP)

The impulse Pass $\rightarrow$ no opposing impulse $\rightarrow$ arrhythmogenic focus

The circle won't accept any impulse from SAN as long as it's faster as any impulse will get there in RP refractory Period.

WhiteKnightLove

INVESTIGATIONS

A) For diagnosis of the TYPE of arrhythmia:

★ 1. ECG (resting & ambulatory). ★

2. EPS (electrophysiologic study).
(detect exact site of electrical focus)
بیشتر تشخیص نوعی مکانیسم
→ needed in electrical ablation

بواسطه قسطره
microelectrodes are inserted
to record electrical activity
V. precisely
→ electrical mapping

B) For diagnosis of the CAUSE of arrhythmia:

★ 1. ECG. ★ [AMI]

2. IMAGING:

- CXR. [RHD]
- Echocardiography [Valvular disease]
- Cardiac catheterization & angiocardiology. [IHD]

3. OTHERS:

- Cardiac enzymes: for AMI.
- Thyroid function tests: for Hyperthyroidism.
- Serum K: for ↓ K or ↑ K.

فهرس

CLINICAL CLASSIFICATION

A) SINUS RHYTHM DISTURBANCES:

- ① 1. Sinus tachycardia.
- ② 2. Sinus bradycardia.
- X 3. Sinus arrhythmia.

B) PATHOLOGICAL TACHY - ARRHYTHMIAS:

- ④ re-entry
- ② 1. Supraventricular tachycardia (SVT).
- ④ 2. Atrial flutter.
- ⑤ 3. Ventricular tachycardia (VT).
- ⑥ 4. Atrial fibrillation (AF).

C) PATHOLOGICAL BRADY - ARRHYTHMIAS:

- ! ① 1. Nodal (Junctional) rhythm.
- ② 2. Heart block.
- X 3. Sick sinus syndrome.

D) OTHERS:

- ④ 1. Premature beats (Extrasystoles).
- X 2. Pre-excitation syndromes.

NB asymptomatic arrhythmias

- ① Sinus brady
- ② Sinus tachy
- ③ 1AVB
- ④ Extrasystole

Irregular

Etiology

Sinus tachy

Sinus Brady

SupraVent. tachy

atrial Flutter

VT

AF

Extrasystoles

Heart Block

Physiologic

- Exercise
- Emotion
- Pregnancy
- Anxiety
- Shock
- Thyrotoxicosis
- Hyperthyroid
- Hypo thyroid
- Hypo vitamin C
- Hypo vitamin B12
- HF
- PE

Pathologic

- Atrial fibrillation
- Hypertension
- Myocardial infarction
- Hypothyroid
- ICT
- Cholestasis

Physiologic

- Athlete
- Sleep

Pathologic

- Hypertension
- Myocardial infarction
- Hypothyroid
- ICT
- Cholestasis

Physiologic

- Congenital AVN
- Hyperthyroidism
- Hypertension
- Hypothyroidism

Pathologic

- Atrial fibrillation
- Hypertension
- Myocardial infarction
- Hypothyroid
- ICT
- Cholestasis

Physiologic

- Emotional stress
- Smoking
- Caffeine
- Tea

Pathologic

- Atrial fibrillation
- Hypertension
- Myocardial infarction
- Hypothyroid
- ICT
- Cholestasis

Physiologic

- Congenital AVN
- Hyperthyroidism
- Hypertension
- Hypothyroidism

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- Symp. Adrenaline
- Xpans: Atropine
- CCB: Nifedipine
- Thyroid hormone
- Nicotine, Alcohol

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- Digitalis

Pharma

- Sympathomimetic drugs

Pharma

- Antiarrhythmic class IC

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Sinus Tachy	Sinus Brady	Supra. Vent. Tachy	Atrial Flutter	VT	AF	Extrasystoles	Heart Block
① QRS :- Rhythm: regular Rate: 100-180/min D: normal ② P wave :- Normal	① QRS :- Rhythm: regular Rate: 50-60/min D: normal ② P wave :- Normal	① QRS :- Rhythm: regular Rate: 120-250 D: normal ② P wave :- atrial: deformed (upright) $\rightarrow$ then QRS Normal: high: inverted before QRS mid: Masked by QRS low: inverted after QRS	① QRS :- Rhythm: regular "fixed" "irregular" "variable" Rate: 150-200 or 75 D: normal ② P wave :- replaced by "Flutter waves" at a rate 250-350/min typically "saw tooth"	① QRS :- Rhythm: regular Rate: 20-250 D: wide ② P wave :- (absent) Normal rate 60-100 Normal shape Comes before, hidden or after QRS (All dissociated)	① QRS :- Rhythm: Marked irregular Rate: 100-160 D: Normal ② P wave :- replaced by "Fibrillation waves" Fibrillation waves $\rightarrow$ irregular vibrations	① QRS :- Rhythm: occasional irregular Rate: Sinus D: supraventricular: Normal vent: wide Followed by (Comp. Bundle) ② P wave :- Comes early Supraventricular: see SVT Vent: no P wave (Ventricular bundle)	1st°: Prolonged "Fixed" PR interval 2nd°: Mobitz I Progressive PR prolongation until a QRS is dropped then PR return to normal duration & sequence repeats QRS: regular "fixed" irregular "variable" slow 30-50 Normal P wave Normal rate 60-100 2, 3 or 4 before QRS Fixed: 2:1, 3:1, 4:1 Variable: changing 2:1 $\rightarrow$ 3:1 $\rightarrow$ 4:1 3rd°: QRS R: Regular R: 30-40/min D: wide P wave Normal rate 100 before, hidden or after QRS (AV dissociation)

ECG

SINUS TACHYCARDIA

disturbed normal automaticity

DEFINITION

- The SAN discharges at a rapid rate > 100 / minute.

ETIOLOGY

Examples Not all Causes

1. Physiological: (No disease) $\rightarrow$ $\uparrow\uparrow$ sympathetic

- **E**xercise.
- **E**motions.
- Pregnancy.
- Infancy.

2. Pathological: (Disease)

- Fever, $\uparrow 1^\circ \sim \uparrow 10$ HR
- Shock, Reflex "Marek's Law"
- Hyperthyroidism, $\rightarrow$ $\uparrow$ chronotropic $\rightarrow$ Hyperdynamic circulation.
- Hypovolemia, $\rightarrow$ Hypotension. $\Rightarrow$ Reflex
- HEART FAILURE. (compensatory)
- PULMONARY EMBOLISM. (esp. medium sized; low CO)
- Anemia. [Hyperdynamic Circulation]
- Myocarditis. (Compensatory tachycardia)

3. Pharmacological: (Drugs)

- Sympathomimetic drugs: e.g. Adrenaline.
- Parasympatholytic drugs: e.g. Atropine.
- CCBs: e.g. Nifedipine. (Reflex due to hypotension)
- Thyroid hormones. (in Myxedema; over dose)
- Nicotine, Caffeine, Alcohol.

$\rightarrow$ atherosclerosis
 $\rightarrow$ Sinus tachy
 $\rightarrow$ toxic effect in DCM

CLINICAL PICTURE

SYMPTOMS

2. Asymptomatic.
3. PALPITATION: awareness of heart beats
 - (4) rapid, (3) regular.
 - (1) gradual onset, (2) gradual offset.
4. Symptoms of: low cardiac output. >160 [↓ diastolic filling]
5. Precipitation of: angina & HF in susceptible patients.

(due to ↓ CO)

→ (1) ↓ CO (2) ↓ diastole : ↓ Coronary filling (3) ↑ demand

SIGNS

1. PULSE:

- Rhythm: regular. (Sinus)
- Rate: 100 – 180 / min.
- Response to carotid massage:
 - Gradual slowing: of the rate.
 - Gradual acceleration: to the original rate on stopping massage.

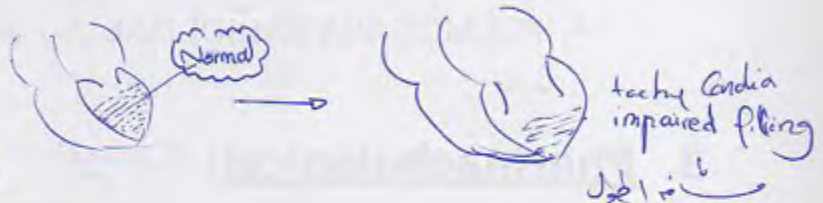
2. NECK VEINS:

- Normal rapid waves with the same rate of pulse.

atrium = Vent. as Source is the same
SAN

3. AUSCULTATION:

- Accentuated S₁.

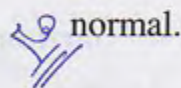


ECG

Normal But Rapid

• QRS: Ventricle

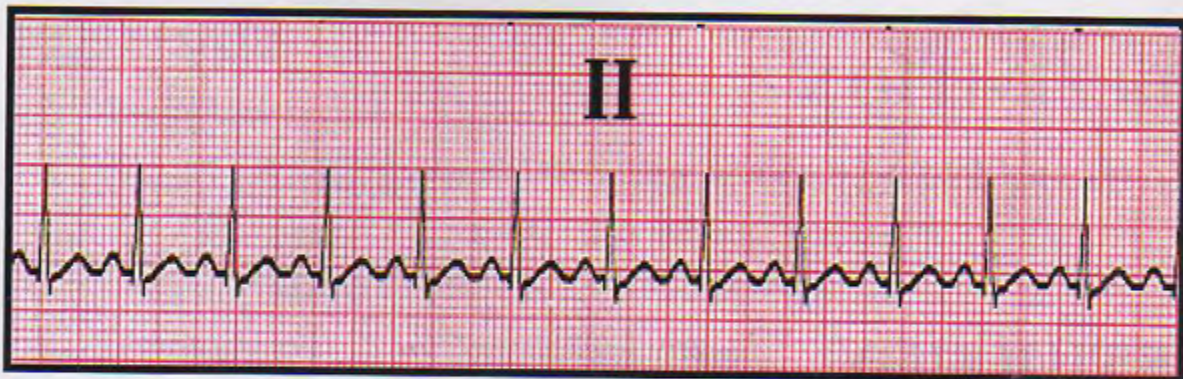
- Rhythm: regular.
- Rate: 100 – 180 / min.
- Duration: normal.



(تسرع قلب طبيعي)

• P wave: atrium

- Normal.



Sinus tachycardia

TREATMENT

1. TTT of the cause, e.g. antithyroid drugs for hyperthyroidism.
2. Sedatives: may be needed. *[may be anxiety & Emotional]*
3. β -blockers: e.g. propranolol, in severe cases.
↓ SAN

SINUS BRADYCARDIA

DEFINITION

- The SAN discharges at a slow rate < 60 / minute.

ETIOLOGY

1. Physiological:

- Athletes.
- Asleep. $\longrightarrow \uparrow$ Parasympathetic

2. Pathological:

- Hypothermia.
- Hypothyroidism.
- Increased vagal stimulation,
- Increased intracranial tension,
- CHOLESTASIS:

$\downarrow$ HR $\downarrow$ BI. Pr
 e.g. Vasovagal syncope.
 e.g. Brain tumours. $\uparrow$ ICT
 Obstructive jaundice.
 $\uparrow$ direct Billirubine
 $\rightarrow$ Brady Cardia due to direct inhibition.

VHC $\rightarrow$ HTN
 $\downarrow$ reflex brady
 CIC $\rightarrow$ direct brady

3. Pharmacological:

- Parasympathomimetic drugs: e.g. Neostigmine.
- Sympatholytic drugs: e.g. β -blockers.
- CCBs: e.g. Verapamil & diltiazem.
- DIGITALIS.

$\rightarrow \ominus$ SAN
 $\rightarrow \oplus$ Vagus

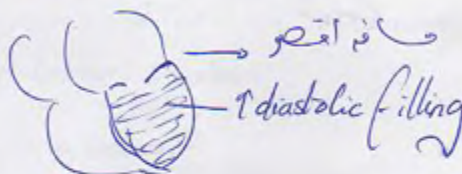
CLINICAL PICTURE

SYMPTOMS

1. Asymptomatic.
2. PALPITATION:
 - ④ slow, *
 - ③ regular.
 - ① gradual onset, ② gradual offset.
2. Symptoms of: low cardiac output.
3. Precipitation of: angina & HF in susceptible patients.

SIGNS

1. PULSE:
 - Rhythm: regular.
 - Rate: * < 60 / minute (50 – 60 / minute, or less).
 - Response to exercise: *
 - Gradual acceleration: of the rate.
 - Gradual slowing: to the original rate on stopping exercise.
2. NECK VEINS:
 - Normal slow waves with the same rate of pulse.
3. AUSCULTATION:
 - Normal or weak S₁.



ECG

Normal Beat Slow

- QRS:
 - Rhythm: regular.
 - Rate: 50 – 60 / min, (or less).
 - Duration: normal.
- P wave:
 - Normal.



Sinus bradycardia

TREATMENT

1. TTT of the cause, e.g. L – thyroxin for hypothyroidism.
2. Atropine: may be needed.

SUPRAVENTRICULAR TACHYCARDIA

DEFINITION

- It is an tachy - arrhythmia originating from above the ventricle.
- SAN is not the pace maker. atrial or Nodal

abnormal automaticity
OR re-entry لاسترجاع

TYPES

1. Atrial tachycardia: the arrhythmia originates from the Atria.
2. Nodal tachycardia: the arrhythmia originates from the AVN. = Functional Tachycardia

ETIOLOGY

- It usually occurs in a normal heart, & therefore its presence does not always denote an ORGANIC HEART DISEASE.

1. Physiological: occurs in a normal heart (no organic heart disease). تحت —> ablation

2. Pathological: (organic heart disease)

- segmental • CAD → ischemia (especially AMI).
- Global • Cardiomyopathy → stretch (and myocarditis). inflammation
- Congenital heart disease.] → Enlarged chambers
- RHD. → F
- Hypertension. → Hypertrophy → ischemia
- Hyperthyroidism. ⊕ excitability (90 → -60)

X • PRE - EXCITATION SYNDROMES: e.g. WPW syndrome.

3. Pharmacological:

- DIGITALIS. ⊕ excitability
 - Sympathomimetic drugs. (e.g. in bronchial asthma) ⊕ excitability
 - Some anti-arrhythmic drugs e.g., class I C. antiarrhythmic
- induce arrhythmia

CLINICAL PICTURE

SYMPTOMS

1. PALPITATION:

④ rapid,

① sudden onset,

③ regular.

③ sudden offset.

→ All re-entry arrhythmias are regular

→ focus

2. Symptoms of:

low cardiac output. (sever in Nodal)

3. Precipitation of:

angina & HF in susceptible patients.

SIGNS

1. PULSE:

- Rhythm: regular.
- Rate: 120 – 250 / minute.
- Response to carotid massage: (Very painful on one side)
- Sudden disappearance: of the arrhythmia may occur.
(Att in hemodynamically stable Pt.)

2. NECK VEINS:

- In atrial tachycardia: normal rapid waves with the same rate of pulse.
- In nodal tachycardia: regular cannon waves with the same rate of pulse.

3. AUSCULTATION:

- In atrial tachycardia: accentuated S₁. (↑ Valvular Component)
- In nodal tachycardia: regular cannon sounds. (↑ Valvular & ↑ Muscular)

ECG

• QRS:

- Rhythm: regular.
- Rate: 120 – 250 / min.
- Duration: normal. 1 + 1/2 → D.D. is Vent. tachy

→ wide QRS

• P wave:

 - In atrial tachycardia:

deformed. (But +ve: upright) : direction from above downwards

 - In nodal tachycardia:

absent or inverted.

High nodal tachy : inverted P wave before QRS

Mid nodal tachy : QRS mask P wave = absent P

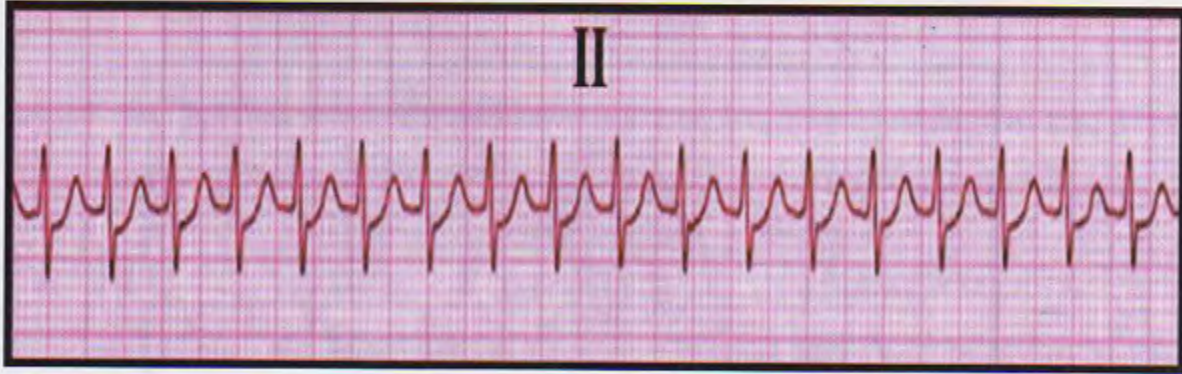
Low nodal tachy : inverted P wave after QRS

White Knight Love

Nodal tachycardia

الاشارة

“Absent p” = Mid Nodal tachycardia.
= Mid Junctional tachycardia.



0.12 - 0.04
0.2 Sec.

Nodal tachycardia

“Inverted p”



“Before QRS” = High Nodal tachy



“After QRS” = Low ~~fast~~ Nodal tachy

TREATMENT

1 During the attack:

A. If the patient is hemodynamically stable:

Slow atrium →

1. Vagal stimulation: carotid sinus massage.

2. Slow the ventricular rate:

- **A**denosine: ↑ Refractory Period of AVN 6 mg IV.
- **β**-blocker (propranolol): 5 mg IV.
- **C**CB (verapamil): 5 mg IV.
- **D**igitalis: ↳ node ↓ rate 1 mg IV.

3. Restore sinus rhythm (Cardioversion): (after ↓ the ventricular rate)

- Chemical cardioversion: Class IA, IC or class III drugs.
- Electrical cardioversion (DC): if chemical cardioversion fails.

↳ Recurrence is Common of the focus (Must prevent future attacks)

B. If the patient is hemodynamically unstable: shock or HF

1. DC cardioversion.

2. Overdrive pacing:

* The atria are paced at a faster rate than the tachycardia rate.

Sudden cessation of pacing is usually followed by:

"restoration of sinus rhythm".

* Recurrence of the focus is Common (Must prevent future attacks)

2 Prevention of a future attack:

A. Maintenance therapy: ↓ excitability

- DRUGS: (Oral), Class IA, IC or class III anti - arrhythmic drugs.

B. Intervention: Need electrophysiologic studies (EPS: Cardiac Mapping)

- Ablation of focus: Catheter (Radiofrequency energy) or surgery.

C. TTT of the cause.

↳ ablation
(Multiple incisions) "like"

ATRIAL FLUTTER

DEFINITION

- A tachycardia in which the atria discharge at a regular rapid rate: 240 – 440 / min.
- A physiological block occurs in the AVN (2:1, or 3:1, or 4:1 block).
- Therefore only $\frac{1}{2}$, or $\frac{1}{3}$, or $\frac{1}{4}$ of the atrial impulses will pass to the ventricles.
- The average ventricular rate will be: $\frac{1}{2}$, or $\frac{1}{3}$, or $\frac{1}{4}$ the atrial rate.
- The block may be:

regular Pulse ← النظم • Fixed (e.g. 2:1), (e.g. 3:1), (e.g. 4:1) or,
 irregular Pulse ← بغير • Variable (e.g. changing from: 2:1 to 3:1 to 4:1.....etc....).

TYPES

1. Type I (common typical): the atrial rate is 240 – 340 / min. $\approx$ 300 / min.
2. Type II (rare): the atrial rate is 340 – 440 / min. $\approx$ 400 / min

ETIOLOGY

- It usually occurs in a patient with ORGANIC HEART DISEASE, However, It may occur in a normal heart.

1. Physiological: may occur in a normal heart (no organic heart disease).
2. Pathological: (organic heart disease)

- CAD (especially AMI).
- Cardiomyopathy (and myocarditis).
- Congenital heart disease.
- RHD.
- Hypertension.
- Hyperthyroidism.
- PRE – EXCITATION SYNDROMES: e.g. WPW syndrome.

3. Pharmacological:

- DIGITALIS.
- Sympathomimetic drugs.
- Some anti-arrhythmic drugs e.g., class I C.

CLINICAL PICTURE

SYMPTOMS

1. PALPITATION:

rapid,

regular (or irregular).

sudden onset,

sudden offset (or $\rightarrow$ AF).

by digitalis ($\oplus$ excitability)

2. Symptoms of:

low cardiac output.

3. Precipitation of:

angina & HF in susceptible patients.

+ Embolization ($\uparrow$ Rate matrium $\rightarrow$ stagnation)

SIGNS

1. PULSE:

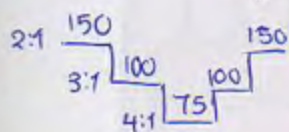
• Rhythm:

regular (fixed block), irregular (variable block).

• Rate:

150 or 100 or 75 / min. (according to AV block).

• Response to carotid massage: ($\uparrow$ AV block from 2:1 $\rightarrow$ 3:1 $\rightarrow$ 4:1)



- Stepwise slowing: of the rate (150 $\rightarrow$ 100 $\rightarrow$ 75 / min).

- Stepwise acceleration: to the original rate on stopping massage.

2. NECK VEINS:

• Multiple "a" waves: **double** or triple or quadruple the rate of pulse.

3. AUSCULTATION:

• Accentuated S₁. (low position of Coaps due to tachy Cardia)

ECG

• QRS:

- Rhythm:

regular (fixed block), irregular (variable block).

- Rate:

150 or 100 or 75 / min. (according to AV block).

- Duration:

normal.

• P wave:

- Replaced by:

multiple "Flutter waves" at a rate of 240 – 340 / min.
 = many P waves

- Typically:

"Saw – tooth appearance".

TREATMENT

1 During the attack:

A. If the patient is hemodynamically stable:

1. Slow the ventricular rate:

it's not anti arrhythmic → **A**denosine: 6 mg IV.

⊙ **β**-blocker (propranolol): 5 mg IV.

⊙ **C**CB (verapamil): 5 mg IV.

it's not anti arrhythmic drug → **D**igitalis: 1 mg IV.

Digitalis, by increasing atrial automaticity, may convert Atrial flutter into AF. If the drug is then stopped, sinus rhythm may be restored.

2. Restore sinus rhythm (Cardioversion): (after ↓ the ventricular rate)

- Chemical cardioversion: Class IA, IC or class III drugs.
- Electrical cardioversion (**DC**): if chemical cardioversion fails.

B. If the patient is hemodynamically unstable:

1. DC cardioversion.

2. Overdrive pacing:

The atria are paced at a faster rate than the tachycardia rate. Sudden cessation of pacing is usually followed by: "restoration of sinus rhythm".

2 Prevention of a future attack:

A. Maintenance therapy:

- DRUGS: (Oral), Class IA, IC or class III anti - arrhythmic drugs.

B. Intervention:

- Ablation of focus: **Catheter** (Radiofrequency energy) or **surgery**.

C. TTT of the cause.

VENTRICULAR TACHYCARDIA

abnormal automaticity
re-entry

DEFINITION

- It is an arrhythmia originating from the VENTRICLE that presents with:
 - Three or more successive ventricular premature beats. *(if not successive = Multiple extrasystoles each beat: QRS prolonged (wide))*
 - Rapid Regular tachycardia at a rate of: 120 – 250 / min. *(if QRS not wide = not ventricular (nodal atrial))*
- Since there is no retrograde conduction in the AVN, there will be AV dissociation:
 - The ventricles will be controlled by the ventricular focus: (VR 120 – 250 / min).
 - The atria will be controlled by the SAN: (AR: 60 – 100 / min).

TYPES

1. Sustained: persists more than 30 sec. or causes hemodynamic instability. *→ (shocked or HF)*
2. Non-sustained: persists less than 30 sec. with no hemodynamic instability.

ETIOLOGY

- It very rarely occurs in a normal heart, & therefore its presence almost always denotes an ORGANIC HEART DISEASE.

1. Pathological:

CAD

- Cardiomyopathy (especially AMI).
- Congenital heart disease. (and myocarditis).
- RHD.

- Hypertension.

- Hyperthyroidism.

- PRE – EXCITATION SYNDROMES: e.g. WPW syndrome.

2. Pharmacological:

- DIGITALIS.
- Sympathomimetic drugs.
- Some anti-arrhythmic drugs e.g., class I C.

CLINICAL PICTURE

SYMPTOMS

1. Palpitation: rapid, regular.
sudden onset, sudden offset.
2. Symptoms of: low cardiac output & shock are more common.
3. Precipitation of: angina & heart failure in susceptible patients.
4. SUDDEN DEATH: if converted to Ventricular fibrillation (VF).

VF

منقذتهن تخلف امق يموت

SIGNS

1. PULSE:

- Rhythm: regular! "re-entry system"
- Rate: 120 – 250 / min.
- Response to carotid massage: no response (no vagal supply to ventricles).

2. NECK VEINS:

- "a" waves: normal rate (60 – 100 / min) & less than the pulse rate.
- OCCASIONAL CANNON WAVES.

AV dissociation
SAN قس سرعة

غلك بالاد

3. AUSCULTATION:

- OCCASIONAL CANNON SOUNDS.
- Variable intensity of S₁. (High & higher → tachycardia)

ECG

• QRS:

- Rhythm: regular.
- Rate: 120 – 250 / min.
- Duration: wide.

• P wave:

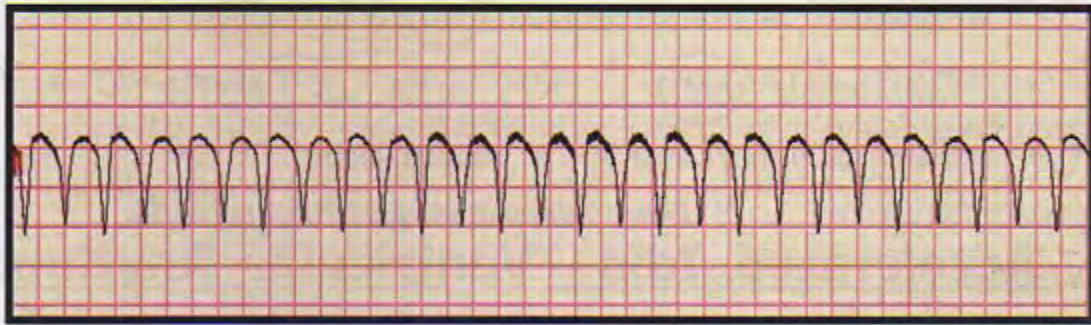
(may not appear) All hidden due to Rapid Rate of QRS

- Normal in rate (60 – 100 / minute) & shape.
- Comes before, after or is hidden by the QRS (AV dissociation).

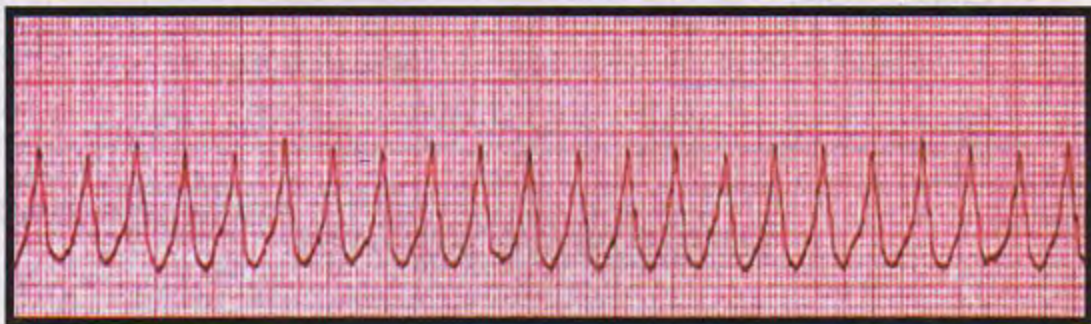
Ventricular tachycardia

QRS wide

+ Regular + Rapid



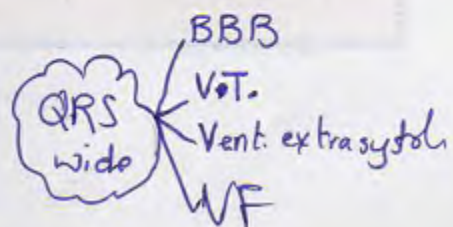
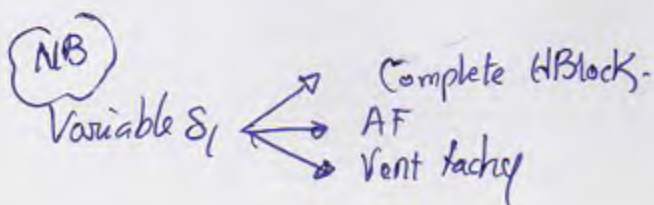
Q waves



R waves



R waves



SPECIAL TYPES OF VT

1. Torsade de pointes:

- QRS complexes change continuously & rapidly & irregularly from an upright to an inverted position (twisting of points)
- Causes include: AMI, $\downarrow K$, $\downarrow Ca$. $\rightarrow [RMP \approx -90 \rightarrow -60]$
- It is serious & may cause VF & sudden death.

2. Accelerated Idio - Ventricular Rhythm:

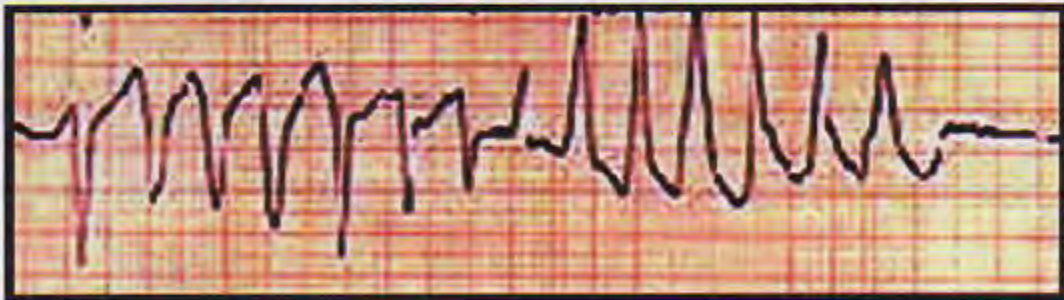
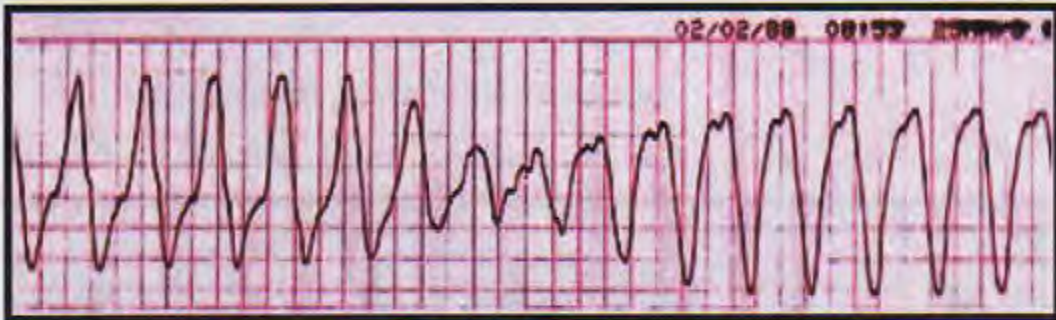
- An ectopic ventricular pacemaker discharges at a rate: 60 - 120 / min, and controls the ventricles only resulting in a slow VT.
- Causes include: AMI, post-coronary thrombolysis (reperfusion arrhythmias).
- It is transient & rarely causes hemodynamic disturbances.

NB transient as when SAN in tachy rhythm $\rightarrow$ faster than pacemaker

NB it's ~~also~~ in AMI & reperfusion: due to accumulation of arrhythmogenic metabolites

Torsade de pointes

Metabolites O_2^+ & K^+ & Ca^{++} reperfusion wash out to all myocardium



Good indicator for Reperfusion (successful)

TREATMENT

1 During the attack:

A. If the patient is hemodynamically stable:

1. Lidocaine: ^{Class Ib} initial bolus of 2 mg / kg IV, followed by maintenance infusion of 1- 4 mg / min. (first drug of choice).
2. Procainamide or Amiodarone: ^{Class Ia} IV (second drug of choice). ^{Class III}
3. Bretylium: ^{Class III} IV (in resistant cases).

B. If the patient is hemodynamically unstable:

- DC cardioversion immediately, followed by IV lidocaine.

2 Prevention of a future attack:

A. Maintenance therapy:

- DRUGS: (Oral), Class I or class III anti - arrhythmic drugs.

B. Intervention:

→ elective not Emergency.

- Ablation of focus: Catheter (Radiofrequency energy) or surgery.

- Implantable Cardioverter Defibrillator (ICD): جهاز بيتزرع في القلب
- "Anti - tachycardia pacing" يمكن يموت في أي لحظة
- ^{يرجع} Sinus rhythm
- ^{ablate} كد لتأجيل
- ^{Focus} أو منشأ ضربات القلب

C. TTT of the cause.

Overdrive pacing - حاجم نري

ATRIAL FIBRILLATION

abnormal automaticity
re-entry

DEFINITION

- A form of tachycardia in which the atria discharge at a rate: 400 – 600 / min.
- An irregular block occurs in the AVN allowing only some impulses to pass to the ventricles in an IRREGULAR manner.

Focus discharge regularly

- Therefore, the ventricular beats will be:

Irregularity in

- Rhythm: markedly irregular. (Very irregular block by AVN)
- Rate: 100 – 160 / min. (heterogenous refractory period)
- Force: "variable".

→ pulse deficit. [Variable filling]

Cardiac Cycle

ETIOLOGY

1. Primary (Idiopathic): "LONE AF" (not secondary to any disease).

- It is more common in old age, and may be slow.

2. Pathological: (Secondary to different diseases)

- ✓✓ CAD (especially AMI).
- Cardiomyopathy (and myocarditis).
- Chest disease, especially COPD. (Hypoxia! 2nd most common arrhythmia in COPD...)
- Constrictive pericarditis. (fibrous → stretch)
- Congenital heart disease, especially ASD.
- ✓✓ RHD, especially MS.
- ✓✓ Hypertension.
- ✓✓ Hyperthyroidism.

- PRE-EXCITATION SYNDROMES: e.g. WPW syndrome.

3. Pharmacological:

✓✓ DIGITALIS.

- Sympathomimetic drugs.
- Some anti-arrhythmic drugs e.g., class I C.

NR

Most Common arrhythmia in COPD is MAT

Multifocal atrial Tachy

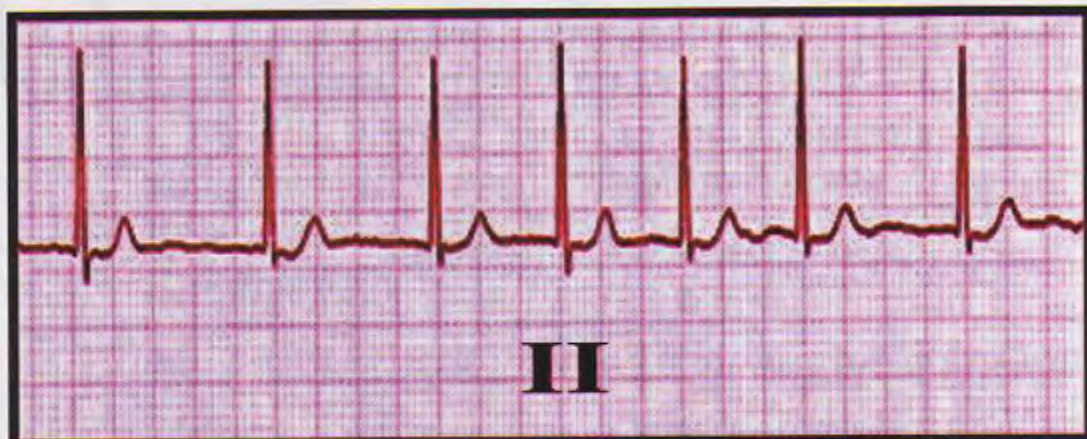
ECG

• QRS:

- Rhythm: markedly **irregular**. *D.D. in Midnodal tachy*
- Rate: 100 – 160 / min. *(except in slow AF.)*
- Duration: normal.

• P wave: **"Absent p"**

- Replaced by: fibrillation waves (irregular vibrations).



NB

Extrasystole

AF

① Rhythm

- occasional irregularity

- Marked irregularity

② pulse deficit

< 10

> 10

③ Exercise

- usually ↓ irregularity

- ↑↑↑ irregularity

- May ↑

- absent a wave

④ Neck v.

- occasional irregularity

⑤ ECG

QRS:

- irregular

QRS

- irregular

- Sinus Rhythm

- 100-160/min (except in slow AF)

- Normal duration or abn.

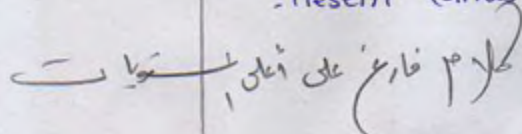
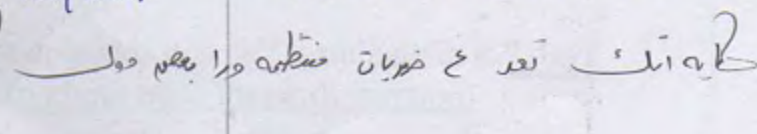
- Normal duration

P wave

- Present (sinus rhythm)

P wave

- Fibrillation waves (absent P)

—  —  —

معظم لائن ليہ انعطاس على العلاج

TYPES

"AHA / ACC / ESC * Classification"

- The ACC, AHA and ESC recommend in their guidelines the following classification system based on: Timing & Termination.

AF Category	Defining Characteristics
First detected (Acute)	only one diagnosed episode (<u>< 48 hours</u> in duration) e.g. in lone AF.
Paroxysmal	recurrent episodes that <u>self-terminate</u> in <u>< 7 days</u> (without need for cardioversion) NB: needs observation & Follow up.
Persistent	recurrent episodes that last <u>> 7 days</u> (<u>not self-terminating</u> ; <u>with need for cardioversion</u> , chemical or electrical cardioversion)
Permanent	long term AF <u>> 7 days</u> , in which cardioversion either <u>failed</u> or was <u>not attempted</u> ! (NB: C.I.: Cardioversion) قاصر سنة سنتين AF خطر ترجيع Sinus

لأنه لانه على thrombi لونه SAN ال atrium يمزب بقوة discharge emboli

- The ACC, AHA and ESC guidelines also describe additional AF categories in terms of: Etiological considerations,

Lone atrial fibrillation (LAF):	Absence of cardiac or any other disease. ^{diagnosed by exclusion}
Secondary AF:	Secondary to different diseases. (to treat cause)
Valvular AF:	Presence of RHD.
Non - valvular AF:	Absence of RHD. ^{ليس انعطاس على Anticoagulant}

* ACC = American College of Cardiology, AHA = American Heart Association,
ESC = European Society of Cardiology

TREATMENT

1 During the attack:

A. If the patient is hemodynamically stable:

(Rhythm Control)

1. Restore sinus rhythm:

"CARDIOVERSION"

- INDICATIONS:

- Recent onset AF: < 12 months duration.
- Absence of: embolization or history of embolization.
- Absence of: atrial thrombus or significant AE.

- CONTRA - INDICATIONS:

- Longstanding AF: > 12 months duration.
- Presence of: embolization or history of embolization.
- Presence of: atrial thrombus or significant AE.

- METHODS:

- Chemical cardioversion: Class IA, IC or class III drugs.
- Electrical cardioversion (DC): if chemical cardioversion fails. (elective)

- PRECAUTIONS:

- Anticoagulation should be given: heparin
 - i. Before cardioversion (3 - 4 weeks).
 - ii. After cardioversion (3 - 4 weeks).

- Digitalis should be stopped before electrical cardioversion (DC). (elective)

2. Slow the ventricular rate:

"RATE CONTROL"

- Indication: when cardioversion fails or is contraindicated.
- Drugs: **B**-blocker (propranolol), **C**CB (verapamil), **D**igitalis. (↑ AV Nodal Block)

B. If the patient is hemodynamically unstable:

1. DC cardioversion.

2. Overdrive pacing:

The atria are paced at a faster rate than the tachycardia rate.
Sudden cessation of pacing is usually followed by:
"restoration of sinus rhythm".

Restore sinus rhythm
العودة الى
السرعة الطبيعية

emboli or Thrombi

emboli or Thrombi

Echo

stroke
peripheral embolism

stretch

will induce serious types of arrhythmias

Vent. rate

[see digitalis]

2 Prevention of a future attack:

A. Maintenance therapy:

- DRUGS: (Oral), Class IA, IC or class III anti - arrhythmic drugs.

B. Intervention:

- Ablation of focus: **Catheter** (Radiofrequency energy) or **surgery**.

C. TTT of the cause. (i.e. no a line in Lone AF)

Cases
Written
Oral
JG

Indications of Anticoagulation in AF

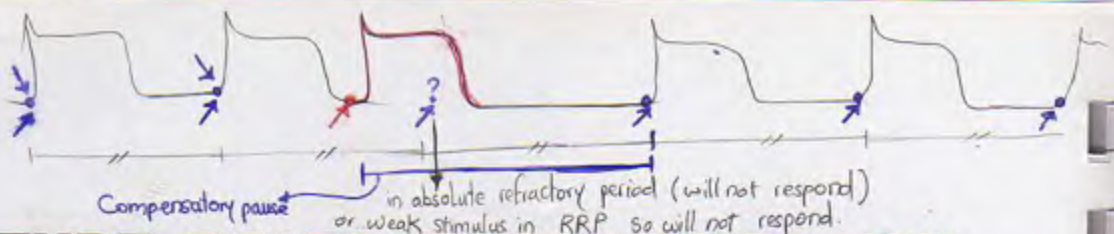
1. Cardioversion (Before & after). [شهر قبل و شهر بعد]
↳ Heparin ↳ no Emboli, no thrombi
2. Embolization or presence of atrial thrombus. (INR: 2.5 - 3.5)
detected by Echo
3. Valvular AF, especially MS. (INR: 2 - 3)
↳ ↑ incidence of thrombosis 40%
4. Non - valvular AF with associated risk factors for thromboembolism:
 • Age > 75 years. (atherosclerosis)
 • Presence of: CAD, HF, HTN.
 • Associated DM. (accelerated atherosclerosis)
 (INR: 2 - 3)

INR
Controlled
By
PC
80-120%
&
PT

CAD → mural thrombi

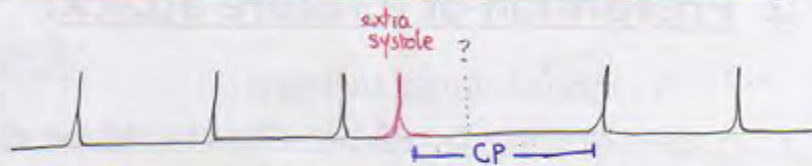
HTN → endothelial dysfunction → accelerate atherosclerosis

HF → Myopathy, Thyrotoxicosis, ...
↳ Systemic Congestion & slow Circulation



PREMATURE BEATS (EXTRASYSTOLES)

DEFINITION



- They are **ectopic** cardiac impulses occurring before the expected sinus impulse causing premature beats.
- When the normal sinus impulse arises, the heart will not respond because it will be in the: "Refractory period". [Compensatory pause]

These **ectopic** cardiac impulses may arise:

1. Supraventricular (from the Atria or AVN) or,
2. Ventricular (from the Ventricles).

ETIOLOGY

1. Physiological: **may** occur in a normal heart (no organic heart disease).

- Emotions. ★
- Excessive: *smoking, coffee, tea.* حركات

2. Pathological: (organic heart disease)

- CAD (especially AMI).
- Cardiomyopathy (and myocarditis).
- Congenital heart disease.
- RHD.
- Hypertension.
- Hyperthyroidism.

● HYPOKALEMIA. (↑Excitability)

1. Pharmacological:

- DIGITALIS.
- Sympathomimetic drugs.
- Some anti-arrhythmic drugs e.g., class I C. Pro-arrhythmia

CLINICAL PICTURE

SYMPTOMS

1. Asymptomatic.
2. PALPITATION:

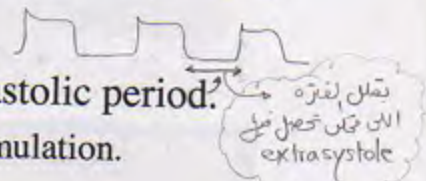
irregular.

مضطربة واحدة بس
Regular Rhythm

SIGNS

1. PULSE:

- Rhythm:
 - Occasional irregularity.
 - Pulse deficit: < 10 beats / minute. "it's not a special character"
- Rate: VARIABLE according to the sinus rhythm (↑, ↓, or normal).
 tachy brady
- Response to exercise: "VARIABLE"
- ↓ irregularity: due to shortened diastolic period.
 لا كثير ←
- ↑ irregularity: due to sympathetic stimulation.



2. NECK VEINS:

- Normal waves with: Occasional irregularity.

3. AUSCULTATION:

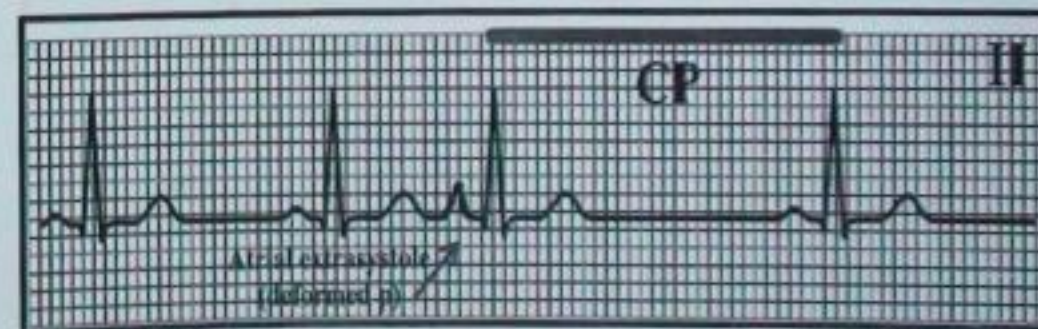
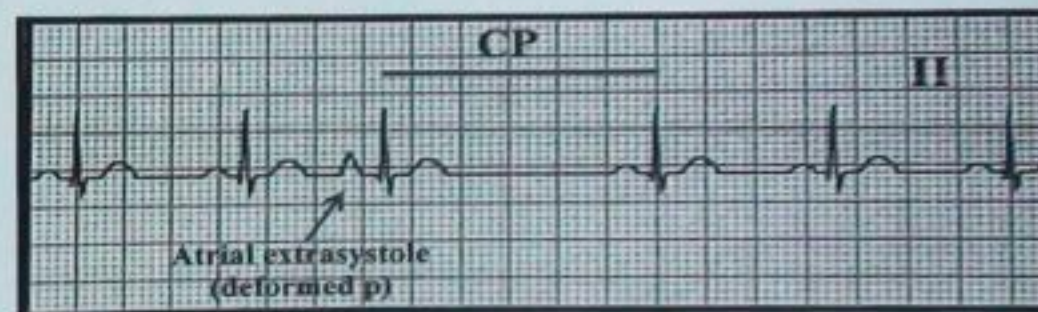
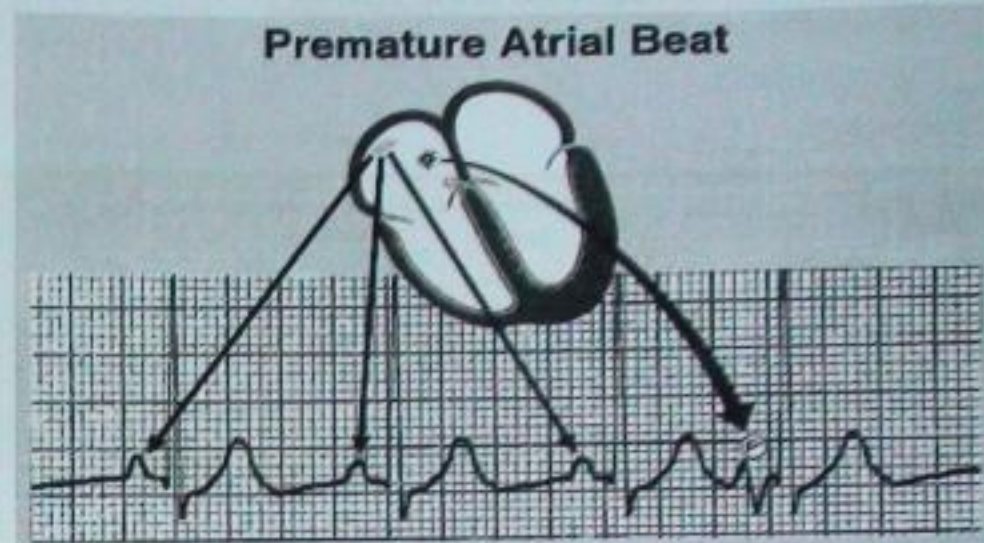
- Normal sounds with: Occasional irregularity.

ECG

- Rhythm: irregular (Occasional irregularity), but: sinus.
 Rate
- The premature beat comes early & is followed by a compensatory pause:
 → than expected beat
- 1. Supraventricular (from the Atria or AVN):
 - Atrial beats: deformed p – wave followed by a normal QRS.
 - Nodal beats: absent or inverted p – wave, a normal QRS.
- 2. Ventricular (from the Ventricles):
 - Ventricular beats: absent p – wave, wide QRS.

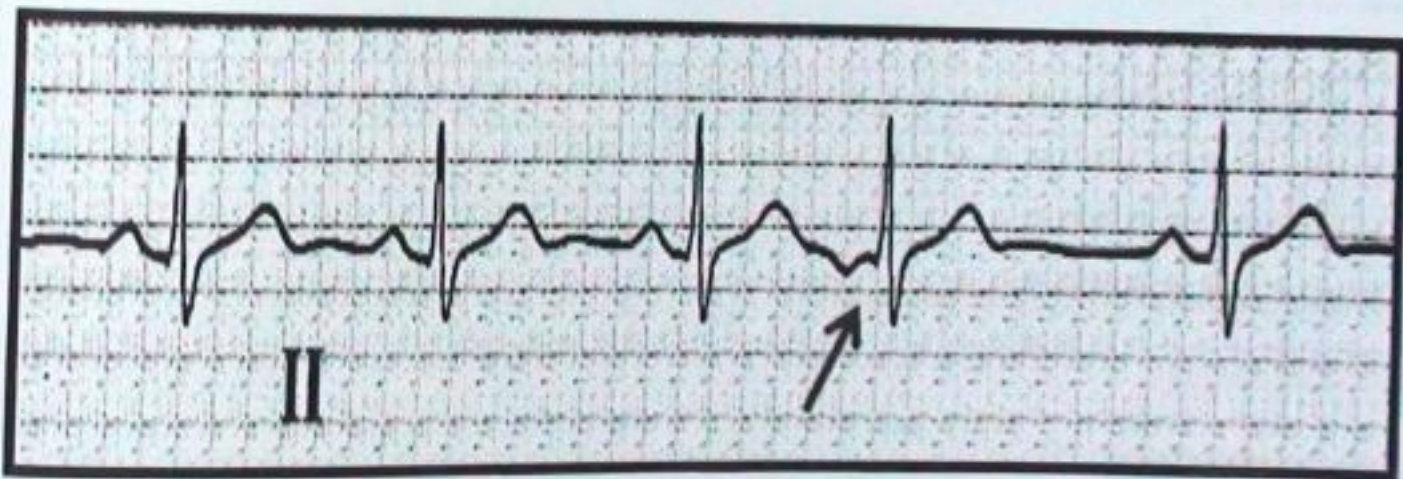
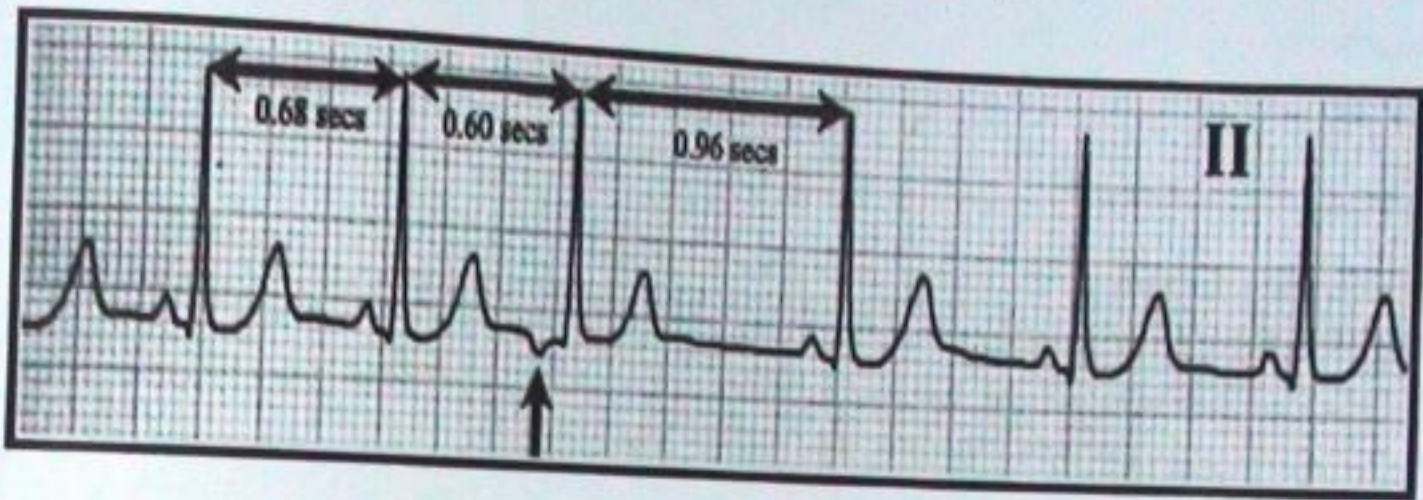
Atrial extrasystole

Premature Atrial Beat

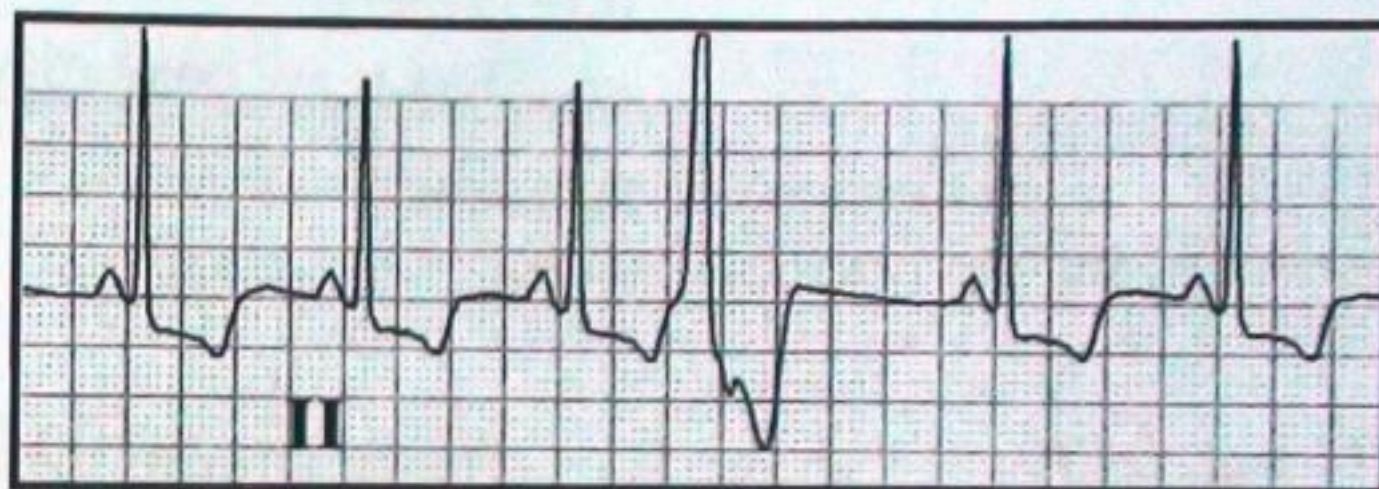


Rhythm	1 up for clinical Extra systoles Most irregular occasional	AF
Pulse Deficit		
exercise		
Neck veins		
ECG		

Nodal extrasystole



Ventricular extrasystole



TREATMENT

Not Serious disease Except Some of Ventricular

1 TTT of the cause.

2 Sedatives:

may be needed.

3 TTT of extrasystoles:

A) Supraventricular beats:

- They usually need no ttt (asymptomatic).
- They may need β -blockers (Symptomatic: causing palpitation).

Sympatholytic (Antiarhythmic)
Anxiolytic

B) Ventricular beats:

- They usually need no ttt (asymptomatic).

التي لا تحتاج علاج - They may need Anti-arrhythmic drugs if they are:

- ① Symptomatic: causing palpitation.

② Multiple. [> 10 in 1min] $\rightarrow$ may become VT, VF in any time

③ Multifocal. $\rightarrow$ VF ("Torsade de pointes" نفس متكررة)

④ Falling on the preceding T-wave (R on T phenomenon).

⑤ Occurring in association with: AMI. $\rightarrow$ Highly excitable [i.e. arrhythmogenic metabolites]

Prophylaxis against arrhythmia

- Anti-arrhythmic drugs are:

ER ① In emergency conditions: e.g. AMI, Digitalis toxicity,

- IV Lidocaine is the drug of choice.

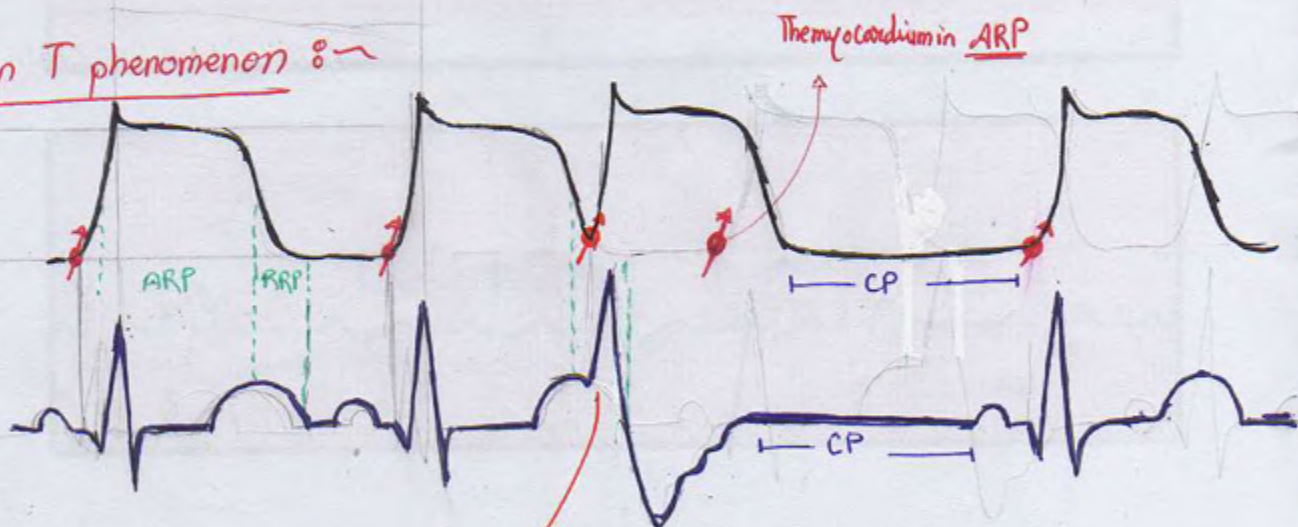
Lidocaine
as antidote
digitalis toxicity

Maintenance ② In stable conditions:

- Oral therapy with class I, II, or III drugs.

Ventricular extrasystole

* R-on-T phenomenon :-



Stimulated the heart in RRP
which means it's a stronger more dangerous focus

May trigger V. serious types of arrhythmia

White Knight Love

NODAL (JUNCTIONAL) RHYTHM

DEFINITION

- An abnormal heart rhythm, where the AVN initiates electrical activity of the heart.
- The impulses spread up & down to activate the atria & the ventricles simultaneously.
- There are 2 possibilities:

1. Nodal tachycardia: “Discussed before”

- Abnormal automaticity in the AVN overtakes the normal SAN.

2. Nodal rhythm: “Discussed below”

- An escape rhythm in which the AVN becomes the pace – maker of the heart discharging at a rate of 40 – 60 / minute, in cases of:
 - o Severe bradycardia: when the SAN discharges at a rate slower than the intrinsic AVN pacemaker.
 - o Heart block: conduction problem between the SAN & the AVN.

ETIOLOGY

- It usually occurs in a patient with ORGANIC HEART DISEASE, However, It may occur in a normal heart.

1. Physiological: may occur in a normal heart (↑↑ vagal tone during sleep).

2. Pathological: (organic heart disease)

- CAD (especially AMI, inferior).
- Cardiomyopathy (and myocarditis).
- SICK SINUS SYNDROME.

3. Pharmacological:

- DIGITALIS.
- β – blockers.

CLINICAL PICTURE

SYMPTOMS

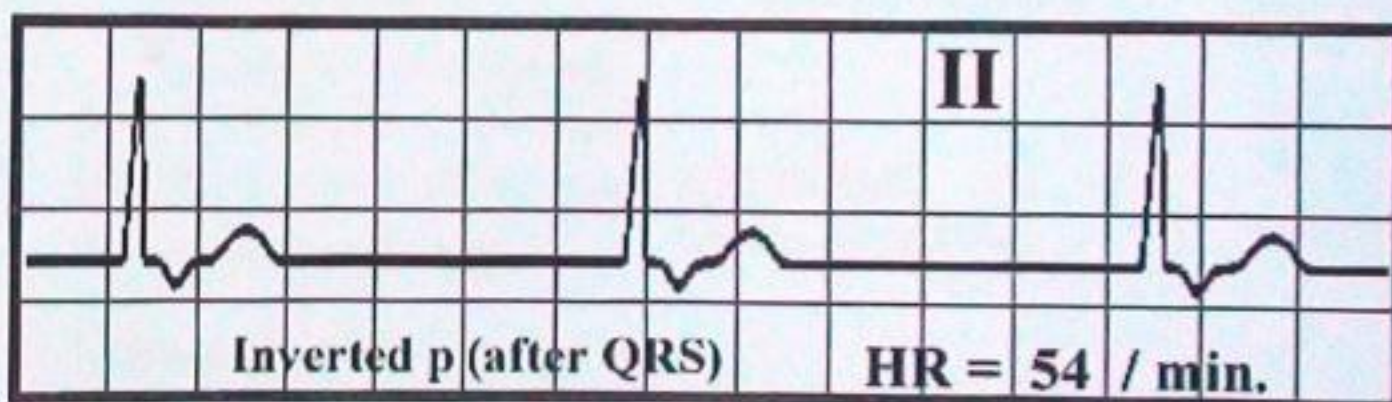
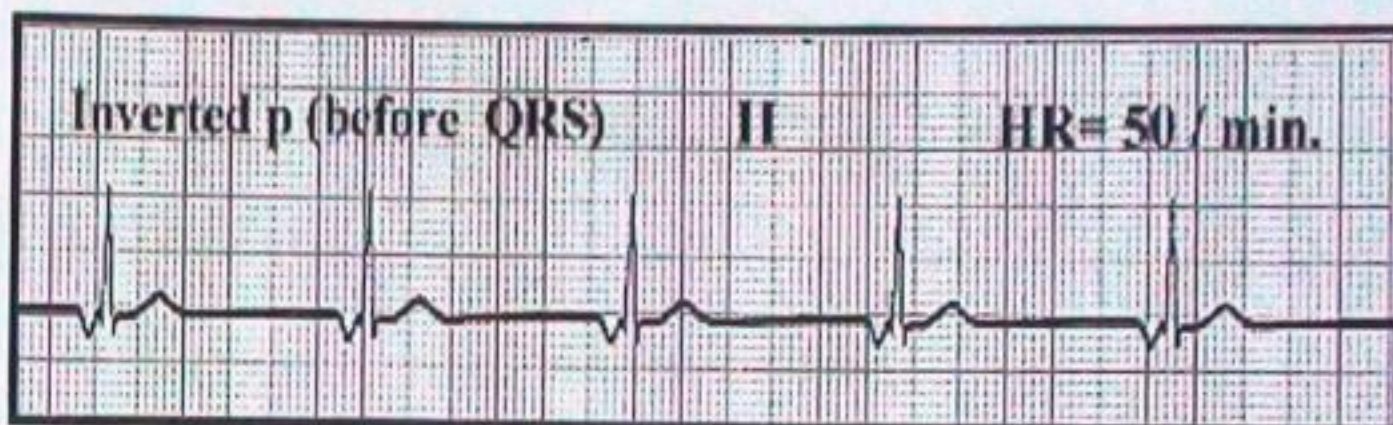
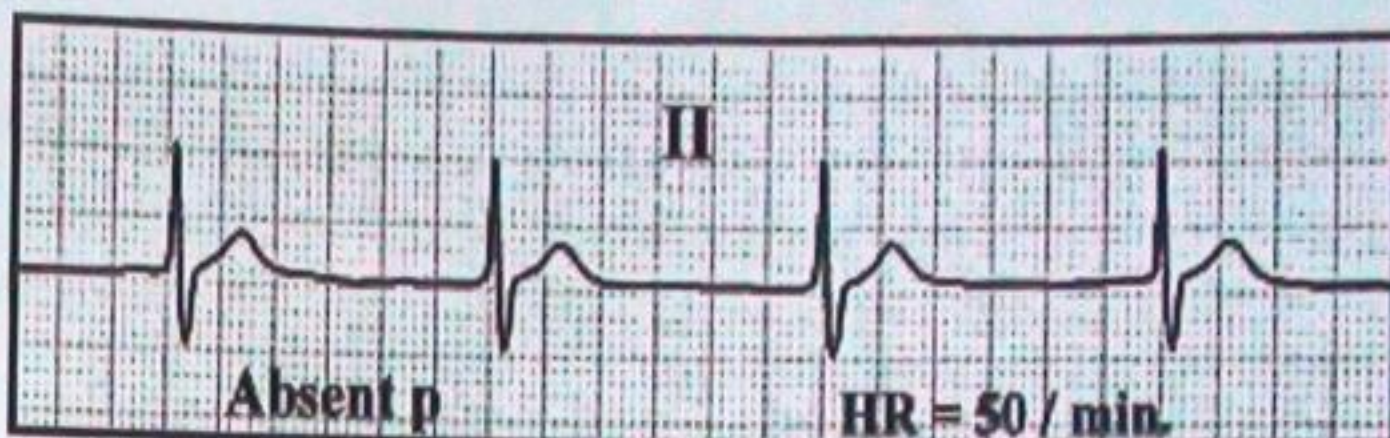
1. Asymptomatic.
2. PALPITATION: regular.
3. Symptoms of: low cardiac output.
4. Precipitation of: angina & HF in susceptible patients.

SIGNS

1. PULSE:
 - Rhythm: regular.
 - Rate: 40 – 60 / minute.
 - Response to exercise:
 - Reversion to sinus rhythm may occur in some cases.
2. NECK VEINS:
 - Regular cannon waves with the same rate of pulse.
3. AUSCULTATION:
 - Regular cannon sounds.

ECG

- QRS:
 - Rhythm: regular.
 - Rate: 40 – 60 / min.
 - Duration: normal.
- P wave:
 - Absent or inverted.



TREATMENT

1. TTT of the cause, e.g. permanent pacemaker for sick sinus syndrome.
2. Atropine: in symptomatic cases.

HEART BLOCK [impaired impulse conduction]

DEFINITION

- A disease in the "Electrical system of the heart":
- Impairment of impulse conduction at any of the following sites:
 1. SAN (Sinoatrial block): between the SAN & the atria.
 2. AVN (AV block): between the atria & the ventricles.
 3. Bundle branch (BBB): along the bundle branches.

AV BLOCK

ETIOLOGY

1. Pathological: (organic heart disease)

- CAD (especially AMI, inferior).
- Cardiomyopathy (and myocarditis).

مشتتوز excitability
نکته عمل NB

Defect in Conductive System →

- Congenital heart disease. "deformity in conductive system"
- Calcific aortic stenosis. → calcific extent to AVN & AV bundle
- Conduction system fibrosis.

Major Criterion "Myocarditis → NB"
Minor Criterion "prolonged PR interval"

• HYPERKALEMIA.

2. Pharmacological: used to protect Ventricle from Atrial Arrhythmias

- **B** - blockers.
- **CCBs**. (Verapamil, Diltiazem.)
- **DIGITALIS**.

TYPES = GRADES = DEGREES

- There are 3 degrees of AV block:

incomplete ↓ digitalis ↓ Complete	1. First degree:	delayed conduction of	<u>ALL impulses.</u>
	2. Second degree:	no conduction of	<u>Some impulses.</u>
	3. Third degree:	no conduction of	<u>ALL impulses.</u>

First degree

DEFINITION

- Delayed conduction of ALL impulses from the atria to the ventricles.
- ALL p waves are followed by a QRS complex.

CLINICAL PICTURE

SYMPTOMS

1. Asymptomatic.
2. Symptoms of: *the cause.*

SIGNS

- No clinical signs.

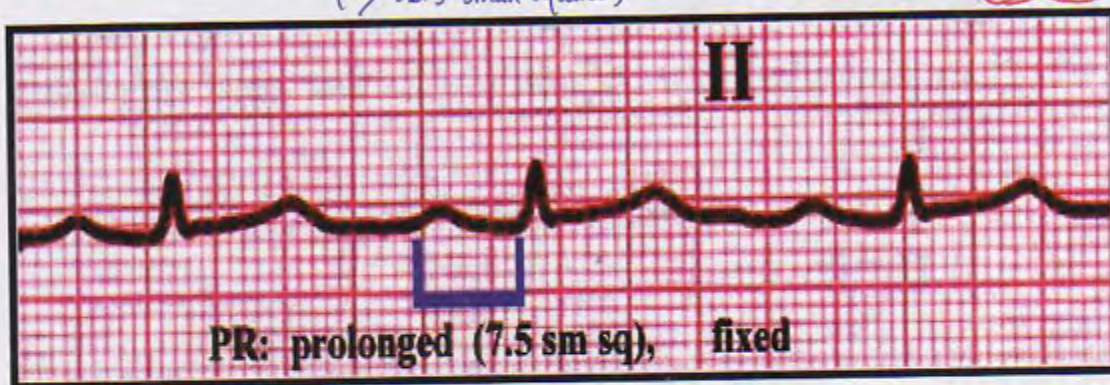
- signs of the *Cause*

ECG

Regular beats

- PR: *prolonged* > 0.2 sec. (> 5 small squares),
($> 3-5$ small squares)

fixed.



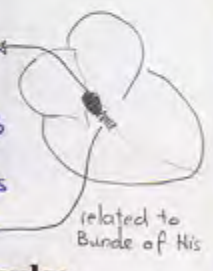
TREATMENT

- TTT of: *the cause.*

Second degree (Incomplete HB)

DEFINITION

- No conduction of Some impulses from the atria to the ventricles.
- Some p waves are not followed by a QRS complex.
- There are 2 types:

1. Mobitz Type I: (Wenckebach phenomenon)¹ (Block is in AVN)
 (PR interval) → 
 - Progressive prolongation of the AV conduction time until conduction fails completely & an atrial impulse is blocked. *dropped QRS*
 ↳ Refractory Period is long enough to prevent atrial impulse to pass
 كل كذا ضرب واحد تقى irregular
 2. Mobitz Type II: (Block is below AVN: Infra-Hisian)
 - Every 2, 3 or 4 atrial impulses, the AVN transmits only one impulse to the ventricles, (so the block will be: 2:1 or 3:1 or 4:1).
 ↳ Refractory Period is long enough to prevent atrial impulse to pass
 كل كذا ضرب واحد تقى regular or irregular
- The block may be:
- Fixed (e.g. 2:1) as Atrial Flutter
 - Variable (e.g. changing from: 2:1 to 3:1 to 4:1.....etc....).
- or,
- Regular ← Irregular

CLINICAL PICTURE

SYMPTOMS

1. Asymptomatic.
2. Symptoms of: the cause.
3. PLUS: In Mobitz Type II,

- PALPITATION:

regular or irregular.

- Symptoms of: low cardiac output.

- Precipitation of: angina & HF in susceptible patients.

as there's severe brady (30-50)

SIGNS

- In Mobitz Type I: irregular pulse, Weak S₁.

¹ The Dutch doctor **Karel Frederik Wenckebach** (1864 - 1940), described the phenomenon.

Mobitz II	Atrial Flutter
* defect in AV bundle	* defect in atrium
* AVN diseased	* AVN protective
* Pulse Rate = sinus (60-100)	* Pulse Rate = (240-340)
* QRS rate: slow (30-50)	* QRS rate (150-100-75)

- In Mobitz Type II:

1. PULSE:

- Rhythm: **regular** (fixed block), irregular (variable block).
- Rate: **slow**, e.g. 30 – 50 / min. (according to AV block).
3:1 2:1

2. NECK VEINS:

- Multiple “a” waves: **double** or triple or quadruple the rate of pulse.

3. AUSCULTATION:

- Weak S1. (*Brady*)

ECG

- In Mobitz Type I:

- PR: Progressive prolongation until a QRS is dropped, **i.e.** until a p wave is not followed by a QRS complex.

Then, the PR interval returns to its normal duration and the sequence is repeated.

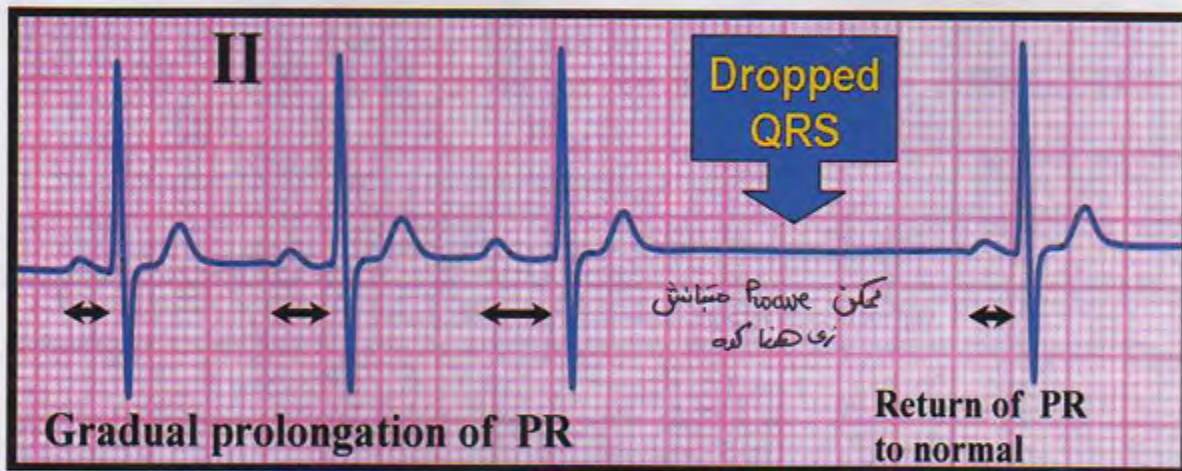
- In Mobitz Type II:

- QRS:

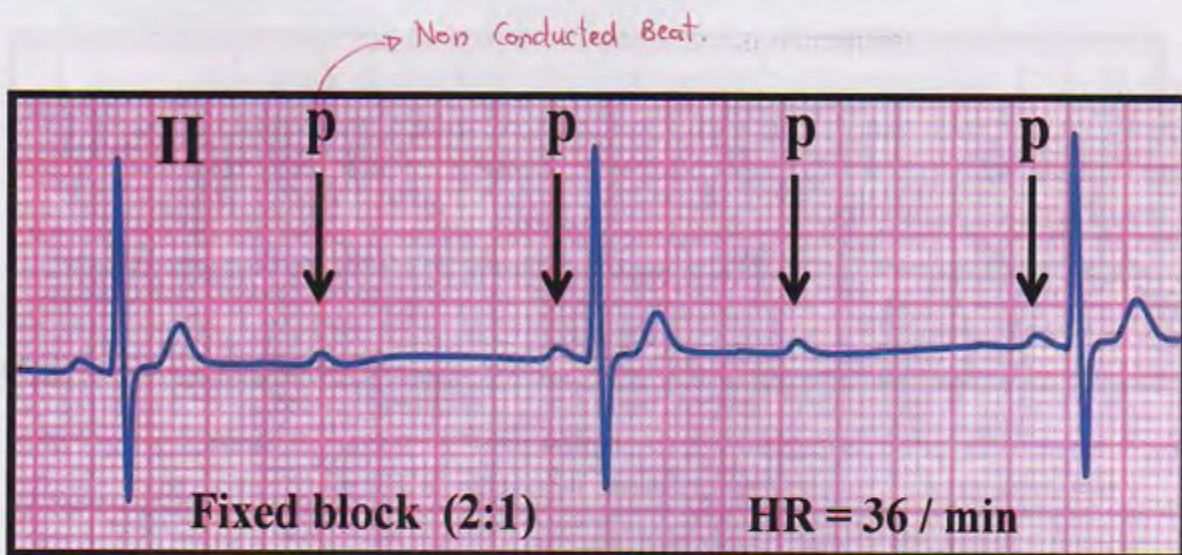
- Rhythm: **regular** (fixed block), irregular (variable block).
- Rate: **slow**, e.g. 30 – 50 / min. (according to AV block).
- Duration: normal.

- P wave:

- Normal rate.
- 2, 3 or 4 p waves occur before each QRS (according to AV block).
- The block may be:
 - o Fixed (e.g. 2:1), **or**,
 - o Variable (e.g. changing from: 2:1 to 3:1 to 4:1.....etc....).



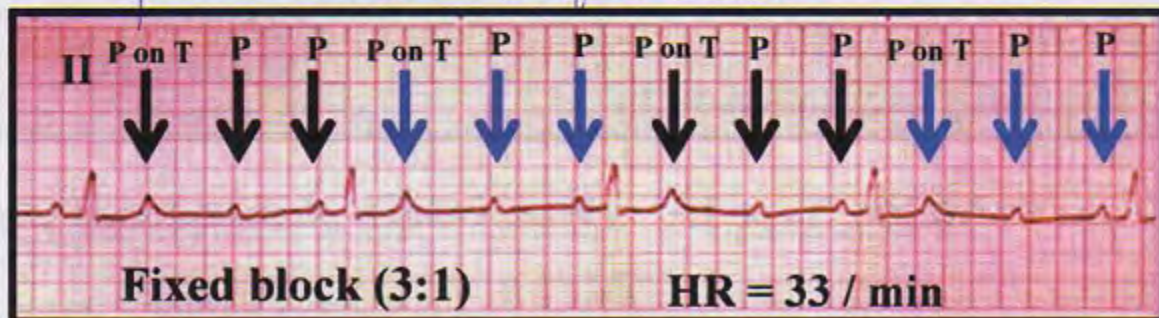
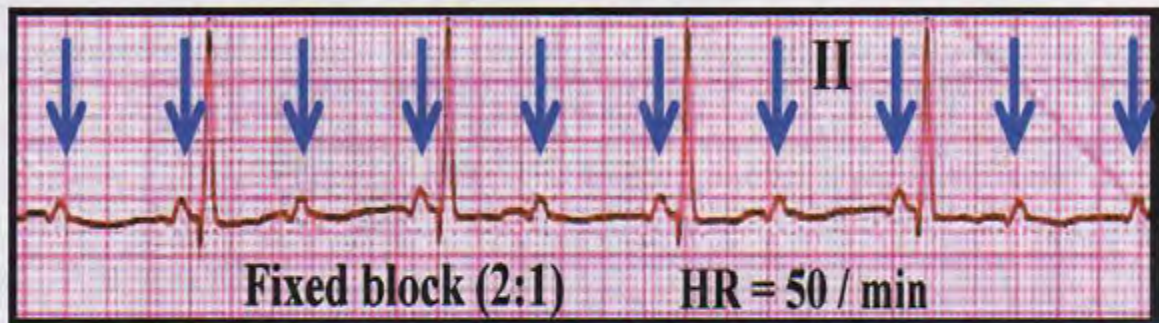
Mobitz Type I: (Wenckebach phenomenon)



Mobitz Type II

Mobitz Type II (Fixed block)

$$\frac{\text{Sinus}}{100} \xrightarrow{2:1} \frac{\text{Vent.}}{50}$$

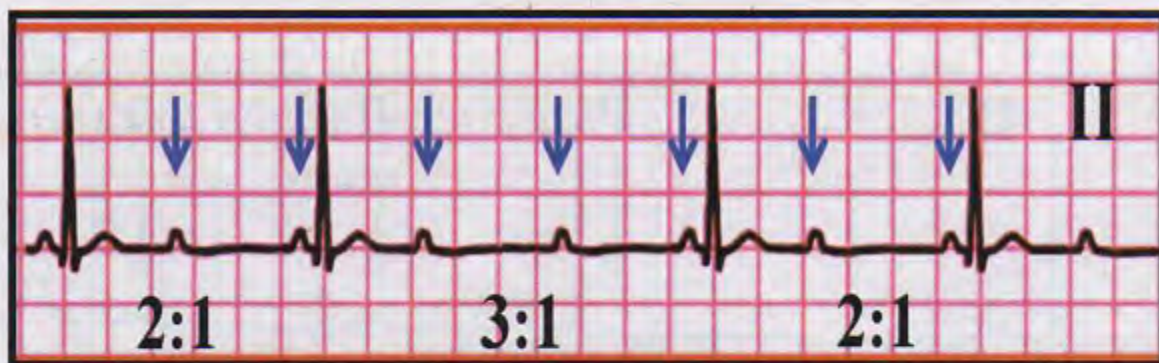


T wave
P wave
P wave regular

atrial contraction on same time of vent. relaxation

$$\frac{\text{sinus}}{100} \xrightarrow{3:1} \frac{\text{Vent.}}{33}$$

Mobitz Type II (Variable block)



TREATMENT

1. TTT of: the cause.
2. Atropine: 1 mg IV. May improve the Conduction
3. PLUS: In Mobitz Type II, Artificial pacemaker.

Ventricular: to avoid severe low CO

Third degree (CHB)

DEFINITION

- No conduction of ALL impulses from the atria to the ventricles.
- How do the ventricles work ??
- They will be controlled by an: *Idioventricular pacemaker*. ^{= Purkinje $\approx$ 30-40}
- No relation between p waves and QRS complexes (*AV dissociation*).
occasional Cannon waves

CLINICAL PICTURE

SYMPTOMS

1. Symptoms of: *the cause.*
2. PALPITATION: *regular.*
3. Symptoms of: low cardiac output.
4. Precipitation of: angina & HF in susceptible patients.
5. *syncope* ADAMS - STOKES ATTACKS: *حزن يوت فيها* $\rightarrow$ if the focus never come back
 - They occur during transmission from one idioventricular focus to another.
 - There is: syncope, cyanosis, with absent pulse, BP & heart sounds.
 - If prolonged: convulsions, coma & death may occur.

SIGNS

1. PULSE:
 - Rhythm: *regular.*
 - Rate: slow, 30 - 40 / min.
2. NECK VEINS:
 - "a" waves: normal rate (60 - 100 / min) & *more* than the pulse rate. $\rightarrow$ VT *فتر*
 - OCCASIONAL CANNON WAVES.
3. AUSCULTATION:
 - OCCASIONAL CANNON SOUNDS.
 - Variable intensity of S₁.

from low $\rightarrow$ High
↓
الاول *↓*
Cannon sounds

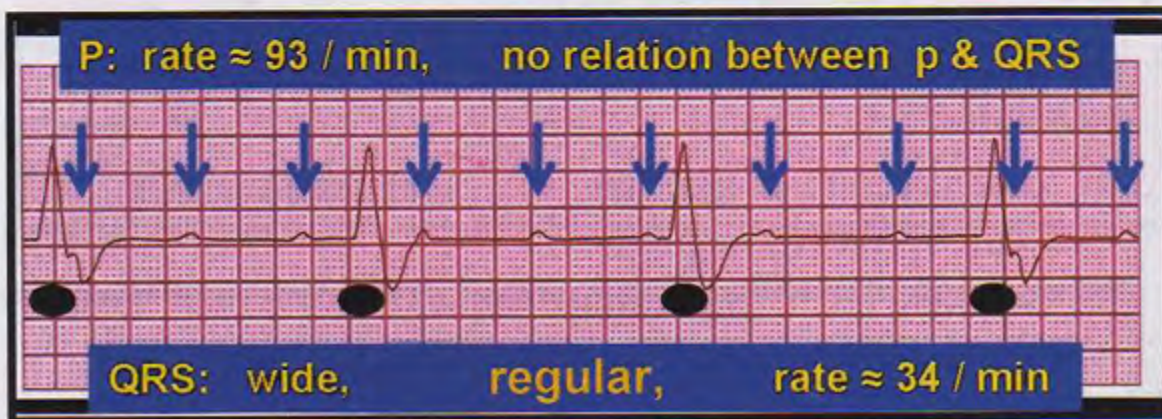
ECG

- QRS:

- Rhythm: regular.
- Rate: 30 – 40 / min. → *معدل 30-40 مع VT*
- Duration: wide.

- P wave:

- Normal in rate (60 – 100 / minute) & shape.
 - Comes before, after or is hidden by the QRS (AV dissociation).
- P faster than QRS*
VT (معدل 30-40)



TREATMENT

1. TTT of: the cause.
2. Atropine: 1 mg IV. *(May improve conduction)*
3. Artificial pacemaker. *(to avoid ADAMS-STOKES attacks & to avoid severe low CO.)*

- Important question: **"Self – assessment"**

"What are the causes of sudden death ??"

"Vogan Williams Classification"

ANTI-ARRHYTHMIC DRUGS

Class	Mode of action	Subclass	Drug	Main uses	Side effects
Class I	Sodium channel blockers slowing the depolarization phase 0 depression	<u>IA</u> Moderate phase 0 depression	<u>Quinidine</u>	Broad spectrum	Idiosyncrasy Cinchonism N. V. Distention <i>auric coating</i>
			<u>Disopyramide</u>	Broad spectrum	Hypotension Anti-cholinergic effects
			<u>Procainamide</u>	Broad spectrum	Hypotension Lupus-like synd.
		<u>IB</u> Minimal phase 0 depression	<u>Lidocaine</u> Epanutin	Ventricular arrhythmias (AMI) (Digitalis toxicity) As lidocaine	Convulsions Confusion Skin rash BM depression Ataxia
		<u>IC</u> Marked phase 0 depression	<u>Propafenone</u>	Broad spectrum	<u>Pro-arrhythmia</u>
Class II	β -blockers		<u>Propranolol</u> & <u>Sotalol</u> <i>↓ excitability ↓ AVN</i>	ST, SVT Atrial flutter AF Extrasystoles	Refer to the chapter of "angina"
Class III	Potassium channel Blockers slowing the repolarization (prolong the RF)		<u>Amiodarone</u> <i>(contain iodine so affect thyroid)</i>	Broad spectrum	Corneal deposits Thyroid problems Pulmonary fibrosis Hepatotoxicity
			<u>Bretylum</u>	Resistant VT	Hypotension
Class IV	<u>CCBs</u>		<u>Verapamil</u> <i>↓ AVN</i>	SVT Atrial flutter AF	Refer to the chapter of "angina"

Drugs with anti-arrhythmic action:

1. Adenosine: it ↓ conductivity in the AV node, so it is indicated in SVT, Atrial flutter.
2. Digitalis: it ↓ conductivity in the AV node, so it is indicated in SVT, Atrial flutter, AF.

they are not
antiarrhythmic
drugs

so not used in Vent. arrhythmias

منخفض التوصيل → Ventricular AF

NBss

Class I: (A, B, C = According to Potency:
 A ± Potent
 B least Potent
 C Most Potent

* Quinidine:
 ↳ idiosyncrasy = unexpected Hypersensitivity reaction
 ↳ Cinchonism = ① Headach ② dizziness ③ Partial deafness ④ Tinnitus

* Disopyramide:
 ↳ -ve inotropic = hypotension as it's anticholinergic
 ↳ Parasympatholytic = Atropine Like = Anticholinergic:
 ① dry mouth ② fever ③ flush ④ Tremors ⑤ ↑IOP ⑥ Urine retention
 (never to be given in Benign prostatic Hyperplasia: BPH)
 never to be given in Glaucoma.

* Procainamide:
 ↳ Lupus Like (as Hydralazine & INH)
 ① Pericarditis ② pleuritis ③ skin rash ④ joint affection
 ↳ Hypotension ? Yes

* Epanutin:
 ↳ Anti epileptic
 ↳ Cerebellar ataxia
 ↳ ⊖ BM
 ↳ skin Rash.

* Propafenone: Marked phase 0 depression = pro-arrhythmic & Anti arrhythmic!

* Pro arrhythmia :- Very rare side effect to an antiarrhythmic drug

Abnormal reaction to a therapeutic dose of an antiarrhythmic drug in the form of production of an arrhythmogenic focus or aggravation of an already present arrhythmia.

من حصول مش هيفي اداء على بطارية () اذ هو لا يوصل الى القلب ارقعه في

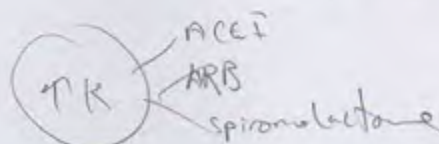
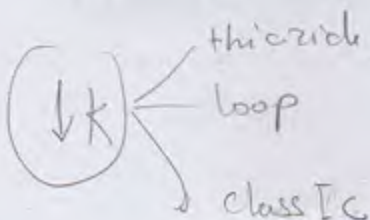


لوصف و كان فيه
 Block
 التام في

الوقت هو اقل
 slow down
 Conduction

انخفاض

arrhythmogenic focus



CARDIOMYOPATHIES

DEFINITION

- Cardiomyopathy, which literally means "Heart muscle disease", is the deterioration of the function of the: MYOCARDIUM.
- A group of disorders that:
 - Primarily involve the: MYOCARDIUM.
 - Usually are: subacute or chronic.
 - Are not the result of: congenital, valvular, pericardial, hypertensive or ischemic diseases.

CLASSIFICATION

1 Etiological Classification:

- Primary: Unknown cause (Idiopathic).
- Secondary: Due to a known cause or Associated with another disease.

2 Clinical Classification:

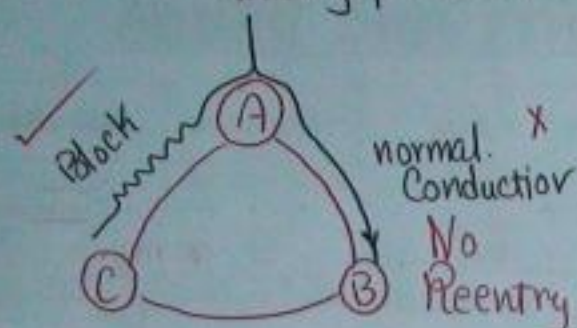
- Dilated (congestive) cardiomyopathy: the most common.
- Hypertrophic cardiomyopathy (HCM): the second common.
- Restrictive (non-dilated, non hypertrophic) cardiomyopathy.

DILATED CARDIOMYOPATHY

ETIOLOGY

- Primary:
 - Idiopathic.
 - Familial.

Proarrhythmia it's an antiarrhythmic drug in a therapeutic dose may induce arrhythmias or aggravates already present arrhythmia.



Rare Proarrhythmia
هذا الدواء يوفى
الشرط الثاني
ويجوز Reentry

- it causes hypo Kalemia as a side effect.

- 1 Thiazide.
- 2 Loop Diuretic.
- 3 propafenone.

how it Treats

- 1 Those S.E is very Rare.
- 2 The drug is very potent
- 3 if these S.E developed you can stop it

b) Secondary:

1. Infective :

- Viral: esp. Coxsackie B virus.
- Others: Bacterial, protozoal.

2. Infiltration & granulomas:

- Amyloidosis, Hemochromatosis, Sarcoidosis.

3. Immunologic: SLE, Scleroderma.

4. Iatrogenic & Toxic: Drugs (Doxorubicin), Toxins (Alcohol).

5. Endocrinal: DM, Acromegaly, Myxoedema.

6. Neurological:

- Duchenne myopathy.
- Friedreich's ataxia.

7. Peripartum cardiomyopathy.

- Important question: "Self – assessment"

"What is meant by: Ischemic cardiomyopathy" ??

PATHOPHYSIOLOGY

- Impaired Systolic function (↓ contractility).
- **Biventricular** dilatation.
 - In approximately 30% of patients with dilated cardiomyopathy:

There is an abnormality in the heart's electrical conducting system (called an "intraventricular conduction delay" or bundle branch block).

CLINICAL PICTURE

- Manifestations of: BIVENTRICULAR FAILURE.
- Manifestations of: COMPLICATIONS:
 - Embolic manifestations: systemic & pulmonary.
 - Arrhythmias, e.g. AF, VT.
 - Sudden death.

INVESTIGATIONS

I. ECG:

- Biventricular enlargement.
- Diffuse nonspecific ST – T changes (*Depressed ST, inverted or flat T*).
- Arrhythmias, *e.g.* (AF, VT).
- Intraventricular conduction delay: BBB.

II. IMAGING:

1. CXR:

- Biventricular enlargement.
- Pulmonary congestion.

2. Echocardiography:

- SIZE: Biventricular dilatation.
- CONTRACTILITY: Impaired global contractile function.

3. Cardiac catheterization & angiocardiology:

- Biventricular dilatation.
- Impaired global contractile function.
- To exclude: CAD.

III. Other investigations for the cause:

- Endomyocardial biopsy: *e.g.* for Infiltration & granuloma.
- Serology: for SLE.
- Thyroid functions: for Myxoedema.

TREATMENT

1. Treatment of: HEART FAILURE *e.g.* ACEIs, β B, Diuretics, CRT.
2. Treatment of: COMPLICATIONS:
 - Anti-coagulant drugs: to prevent embolization.
 - Anti-arrhythmic drugs: for arrhythmias.
 - Cardiac transplantation: in refractory cases.
3. Treatment of: CAUSE: *e.g.* Immunosuppressive drugs in SLE.

HYPERTROPHIC CARDIOMYOPATHY

ETIOLOGY

a) Primary:

- Idiopathic.
- Familial.

b) Secondary:

- Pheochromocytoma.

PATHOPHYSIOLOGY

- ASYMMETRIC LV HYPERTROPHY (SWT > FWT), leading to:
 - Impaired Diastolic function ($\downarrow$ compliance) $\rightarrow$ pulmonary congestion.
 - Obstruction of the LV outflow tract, causing low CO.
 - The septum may bulge into the RV cavity, causing giant a wave.

CLINICAL PICTURE

Symptoms

- Symptoms of: pulmonary congestion.
- Symptoms of: low CO.

Signs

General:

- Pulse: Jerky (sharply rising).
- Neck veins: giant a wave.

Cardiac:

A) Precordial examination:

- Signs of LV enlargement.

B) Auscultation:

- S₄: over the apex.
- Systolic ejection murmur: along the sternal border.
- Pansystolic murmur due to MR: over the apex.

COMPLICATIONS

1. Arrhythmias, *e.g.* AF, VT.
2. Sudden death: probably due to Ventricular arrhythmias.

INVESTIGATIONS

I. ECG:

- LV enlargement.
- Diffuse nonspecific ST – T changes (*Depressed ST, inverted or flat T*).
- Arrhythmias, *e.g.* (*AF, VT*).
- Abnormal Q waves: in inferior leads or lateral chest leads.

II. IMAGING:

1. CXR:

- LV enlargement.
- Pulmonary congestion.

2. Echocardiography:

- SIZE: Asymmetric LVH (SWT > FWT).
- CONTRACTILITY: ↑ contractile function of the LV.

3. Cardiac catheterization & angiocardiology:

- Usually not required: because non-invasive methods are diagnostic.
- ↑ LVDP: due to ↓ LV compliance.

TREATMENT

1. Treatment of: HEART FAILURE But: Avoid positive inotropics.
2. Treatment of: COMPLICATIONS:
 - Anti-arrhythmic drugs: *for arrhythmias.*
 - ICD: *to prevent sudden death.*
3. CCBs (Verapamil) or BB (Propranolol): to relax the septum.
4. SURGICAL TTT: resection of the hypertrophied septum.

RESTRICTIVE CARDIOMYOPATHY

ETIOLOGY

a) Primary:

- Endomyocardial fibrosis.
- Endomyocardial eosinophilic disease (Löffler's endocarditis).

b) Secondary:

1. Infiltration & granulomas:

- Amyloidosis, Hemochromatosis, Sarcoidosis.

2. Immunologic:

- Scleroderma.

PATHOPHYSIOLOGY

- MYOCARDIAL FIBROSIS OR INFILTRATION, leading to:

- The **RV** is involved more than the LV.
- Impaired Diastolic function (↓ compliance) → Venous congestion.
- Mildly Impaired Systolic function (↓ contractility) → Low CO.
- The condition functionally resembles: Constrictive Pericarditis.

CLINICAL PICTURE

Symptoms

- Symptoms of: systemic congestion.
- Symptoms of: low CO.

Signs

General:

- GENERAL SIGNS OF SYSTEMIC CONGESTION.
- GENERAL SIGNS OF Low CO.

Cardiac:

A) Precordial examination:

- Signs of RV enlargement: late & mild.

B) Auscultation:

- S₄ over the tricuspid area & over the apex.

COMPLICATIONS

- Embolic manifestations: systemic & pulmonary.
- Arrhythmias, e.g. AF, VT.

INVESTIGATIONS

I. ECG:

- Low voltage.
- Diffuse nonspecific ST – T changes (*Depressed ST, inverted or flat T*).
- Arrhythmias, e.g. (*AF, VT*).

II. IMAGING:

1. CXR:

- RV enlargement: late & mild.

2. Echocardiography:

- SIZE: Symmetrical myocardial thickening.
- COMPLIANCE: Impaired global ventricular filling.

3. Cardiac catheterization & angiocardiology:

- Square root sign: "Early diastolic dip followed by a plateau" in: RV & LV ventricular pressure tracings.

III. Other investigations for the cause:

- Endomyocardial biopsy: e.g. for Infiltration, granuloma, fibrosis.

DIFFERENTIAL DIAGNOSIS

"Constrictive pericarditis"

	RCM	CP
PERICARDIAL KNOCK	Absent	May be present
PERICARDIAL CALCIFICATION	Absent	May be present
Cardiomegaly	Late & mild	Absent
CT & MRI	-----	Pericardial thickening
ENDOMYOCARDIAL BIOPSY	Diagnostic	-----
Exploratory thoracotomy	Diagnostic	Diagnostic

TREATMENT

1. Treatment of: HEART FAILURE *e.g.* ACEIs, Diuretics.
2. Treatment of: COMPLICATIONS:
 - Anti-coagulant drugs: *to prevent embolization.*
 - Anti-arrhythmic drugs: *for arrhythmias.*
 - Cardiac transplantation: *in refractory cases.*
3. Treatment of: CAUSE: *e.g. Immunosuppressive drugs in Scleroderma.*

CARDIOMYOPATHIES

DEFINITION

- A group of disorders that:

- Primarily involve the: MYOCARDIUM.
- Are not the result of:
congenital, valvular, pericardial, hypertensive or ischemic diseases.

DILATED CARDIOMYOPATHY

ETIOLOGY

a) **Primary:** Idiopathic, familial.

b) **Secondary:**

1. **Infective:** esp. *Coxsackie B virus*.
2. **Infiltration & granulomas:** Amyloidosis, Hemochromatosis, Sarcoidosis.
3. **Immunologic:** SLE, Scleroderma. (anticancer cytotoxic)
4. **Iatrogenic & Toxic:** Drugs (Doxorubicin), Toxins (Alcohol).
5. **Endocrinal:** DM, Acromegaly, Myxoedema.
6. **Neurological:** Duchenne myopathy, Friedreich's ataxia.
7. **Peripartum cardiomyopathy.** (during pregnancy)

3 Causes of deposition of strange material in Myo Cardium

PATHOPHYSIOLOGY

- Impaired **Systolic** function ($\downarrow$ contractility). (exceeded limits of Starling law)
- **Biventricular** dilatation.
 - In approximately 30% of patients with dilated cardiomyopathy:
There is an abnormality in the heart's electrical conducting system (called an "intraventricular conduction delay" or bundle branch block).

CLINICAL PICTURE

- Manifestations of: **BIVENTRICULAR FAILURE.** (R+ + Lt)
- Manifestations of: **COMPLICATIONS:**
 - *Embolic manifestations:* systemic & pulmonary. (stasis)
 - *Arrhythmias,* e.g. AF, VT. (stretch)
 - *Sudden death.* (Fatal vent. arrhythmias)

INVESTIGATIONS

I. **ECG:**

Intraventricular conduction delay: BBB.

II. **Echocardiography:**

- **SIZE:** Biventricular dilatation.
- **CONTRACTILITY:** Impaired global contractile function. (EF, SF)

III. **Other investigations for the cause:** e.g. serology for SLE.

TREATMENT

1. Treatment of: **HEART FAILURE** e.g. ACEIs, β B, Diuretics, CRT.
2. Treatment of: **COMPLICATIONS:** e.g. Anti-arrhythmic drugs for arrhythmias.
3. Treatment of: **CAUSE:** e.g. Immunosuppressive drugs in SLE.
4. Cardiac transplantation: in terminal refractory cases.

NYHA II or III

indication of BB in Cardiology :

- ① HF
- ② MVP
- ③ Fallot's tetralogy
- ④ angina, infarction
- ⑤ HTN
- ⑥ ~~Diabetic~~ Hypertrophic Cardiomyopathy
- ⑦ Antiarhythmic

HYPERTROPHIC CARDIOMYOPATHY

ETIOLOGY

- a) **Primary:** Idiopathic, familial.
b) **Secondary:** Pheochromocytoma.

PATHOPHYSIOLOGY

- Don't affect R.V. & (septal wall thickness) > Free WT in Lt Vent.
- **ASYMMETRIC LV HYPERTROPHY** (SWT > FWT), leading to:
 - Impaired **Diastolic** function ($\downarrow$ compliance) $\rightarrow$ **pulmonary congestion**. (back P. from Lt. Ventr.)
 - Obstruction of the LV outflow tract, causing **low CO**. (relative A.S.)
 - The septum may bulge into the RV cavity, causing **giant a wave**.

CLINICAL PICTURE

- Symptoms: pulmonary congestion, low cardiac output. $\nearrow$ strong Vent. Contract =
- General signs: giant a wave, jerky pulse. "rapid arterial distention" & But low pulse Volume
- Cardiac signs: LVH, systolic ejection murmur along the sternal border. (sub valvular A.S.)
- Complications: arrhythmias (e.g. AF, VT), sudden death. $\nearrow$ $\nwarrow$ ischemia $\nwarrow$ low CO $\nwarrow$ hypertrophy $\nwarrow$ VF

INVESTIGATIONS

- **SIZE:** Asymmetric LVH (SWT > FWT).
- **CONTRACTILITY:** $\uparrow$ contractile function of the LV.

TREATMENT

1. Treatment of: **HEART FAILURE** but: avoid positive inotropics. [relative C. [to digitalis]]
2. Treatment of: **COMPLICATIONS:** e.g. Anti-arrhythmic drugs for arrhythmias.
3. CCBs (verapamil) or $\beta\beta$ (Propranolol): to relax the septum.
4. Surgical ttt: resection of the hypertrophied septum.

[DD Constrictive Pericarditis]

RESTRICTIVE CARDIOMYOPATHY

ETIOLOGY

- a) **Primary:** Endomyocardial fibrosis, Endomyocardial eosinophilic disease (Löffler's endocarditis).

- b) **Secondary:**

1. **Infiltration & granulomas:** Amyloidosis, Hemochromatosis, Sarcoidosis. (ترسب مادة غريبة)
2. **Immunologic:** Scleroderma.

PATHOPHYSIOLOGY

"MYOCARDIAL FIBROSIS OR INFILTRATION"

- The **RV** is involved more than the LV.
- Impaired **Diastolic** function ($\downarrow$ compliance) $\rightarrow$ **Venous congestion**.
- Mildly Impaired **Systolic** function ($\downarrow$ contractility) $\rightarrow$ **Low CO**.
- The condition functionally resembles: **Constrictive Pericarditis**. (square root sign)

CLINICAL PICTURE

- Manifestations of: systemic congestion, low cardiac output.
- Manifestations of: **COMPLICATIONS:**
 - Embolic manifestations: systemic & pulmonary. (rough surface) : fibrotic
 - Arrhythmias, e.g. AF, VT.

INVESTIGATIONS

- I. **Cardiac catheterization & angiocardiology:** "square root sign"
- II. **Investigations for the cause:** Endomyocardial biopsy: e.g. for Infiltration, granuloma, fibrosis.